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PROTRUSION OF THE LOWER LUMBAR INTERVERTEBRAL DISKS*

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THE first published report of a case operated on for protrusion of the intervertebral disk into the spinal canal with which we are familiar is that of Oppenheim and Krause[1] in 1909. Their case was one of a complete transverse lesion of the cauda equina at the third lumbar disk. The lesion was found at operation and removed, and the patient recovered. Middleton and Teacher[2] in England and Goldthwait[3] in America reported cases in 1911. Numerous reports of so-called "enchondroma" appeared between that time and 1929. In the latter year Dandy[4] reported 2 lumbar cases and recognized the distinct character of the lesion, comparing the extruded portion of the disk to the free fragments found in the knee joint and elsewhere. Peet and Echols[5] also recognized the lesion. All these earlier reports had to do with cases showing more or less complete transverse lesions. Schmorl[6] recognized protrusions from the disk margin as of common occurrence, but did not consider them of great neurological significance.

Our own interest was aroused by the case of a young man seen in 1932 with severe intractable sciatica following a fall. Neurologic signs were absent; nevertheless, this patient had a definite protrusion from the disk margin causing compression of the fifth lumbar root. This case and others impelled us[7] to review our cases of enchondroma, and we found that about 75 per cent of the cases were not tumors but were protrusions of normal disk cartilage. In this paper, published in 1934, we called attention to the fact that unilateral leg pain was frequently the outstanding symptom. Since then a very considerable literature on the subject has accumulated. We shall not attempt to enumerate these articles, but instead refer the reader to Saunders and Inman's[8] review.

Protrusion of the intervertebral disk may occur at any level in the spinal canal, but it is only common in the lower two disks, that between the fourth and fifth lumbar vertebrae and that between the fifth lumbar and the sacrum. Over 90 per cent of the protrusions that cause symptoms occur in one or the other of these two disks. We propose to discuss only these lumbar cases.

An examination of the normal fourth lumbar intervertebral disk and the structures behind it reveals that the disk is shaped like a closed horseshoe and that the weakest point is laterally placed. The fifth lumbar root is far out to the side in its sheath. Ventral to the root is the posterior longitudinal ligament, with the disk margin in front of that structure. Posterior to it lie the outer margin of the ligamentum flavum and the inner lip of the articular facet arising from the fifth lumbar vertebra. The nerve lies in a bed of loose connective tissue. Any posterior displacement of or protrusion from the disk margin at this point tends to force the nerve back against the immovable margin of the articular facet (Fig. 1).

The extrusion is almost always laterally placed (Table 1). The extruded fragment is a hard, crumpled mass of fibrocartilage, usually lying free but sometimes attached to the disk margin. It may vary in size from a fragment 2 to 3 mm. in diameter up to a mass large enough to block completely the spinal canal. The mass may lie anterior to the posterior longitudinal ligament of the spine, or the ligament may have been split, allowing the mass to lie in direct contact with the dura. Rarely the extruded mass is soft and is surrounded by a certain amount of gelatinous material, probably from the nucleus.

We wish to emphasize at this point that a large proportion of the cases of sciatica recover spontaneously or under conservative orthopedic

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treatment. No patient should be investigated as a suspected case unless his symptoms have been severe and disabling and have persisted for months rather than weeks.

The symptoms and signs of protrusion of the ening of the ligamentum flavum or arachnoiditis may give essentially the same clinical history and findings on physical examination as does protrusion of the posterior disk. This fact is of no great importance, however, as any two or all three of

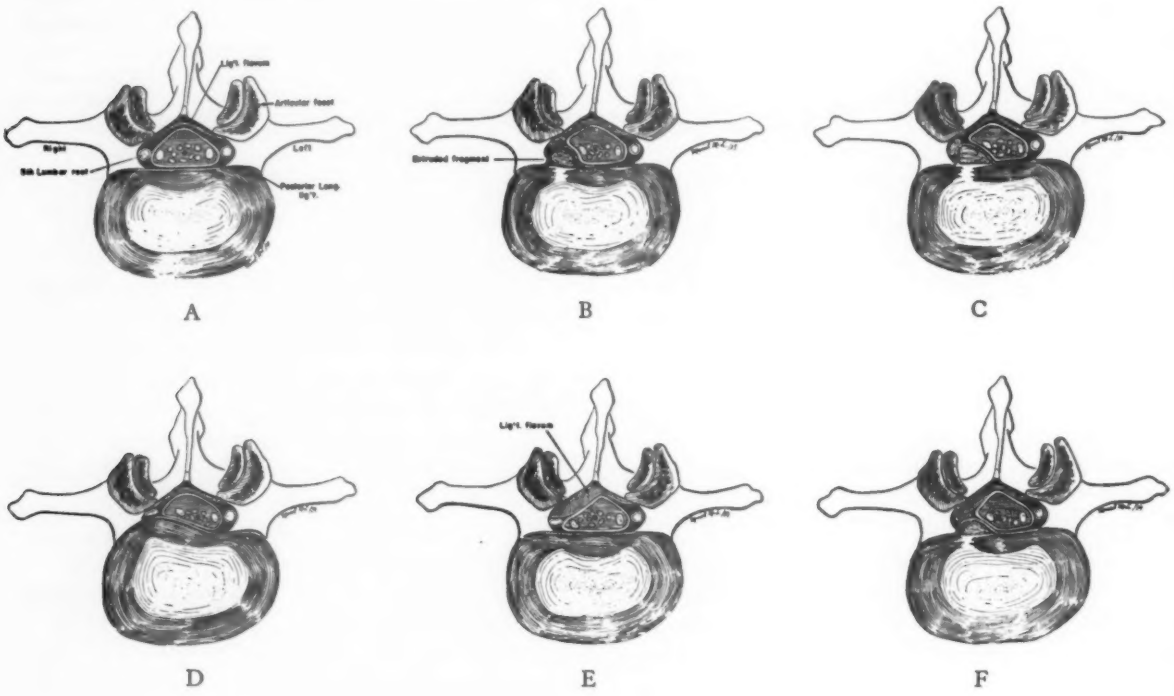


FIGURE 1. *The Lumbar Intervertebral Disk and the Ligamentum Flavum.*

A represents a section through a normal fourth lumbar disk; B, a free fragment of the disk, with compression of the fifth lumbar root; C, a free fragment, with lateral displacement of the fifth lumbar root; D, generalized posterior bulging of the disk; E, a thickening of the ligamentum flavum; F, a thickened ligamentum flavum, with an extended fragment of the disk.

fourth and the fifth lumbar disk are very similar (Table 2). Spurling and Bradford[9] have analyzed their cases and believe that differentiation

TABLE 1. *Incidence of Location and Character of Lesion in Relation to Anatomic Type.*

LESION	INCIDENCE		
	FOURTH LUMBAR DISK	FIFTH LUMBAR DISK	TOTAL
	%	%	%
Location			
Right	30	20	50
Left	22	18	40
Median	9	1	10
Character			
Free fragment	40	28	68
Attached fragment	12	8	20
Posterior bulge	9	3	12

is possible. So far we have been unable to achieve this result in our own cases. This matter is under investigation in various clinics, and it is to be hoped that their views will be confirmed. Thick-

these lesions may be present in the same case, and all of them demand surgical investigation. Rupture of the intervertebral disk probably occurs at least ten times as frequently as do the other two together.

The protrusion may press straight back against the nerve root, or it may lie in the angle between the dural sac and the root sheath. More rarely there is no rent in the annulus but only a more or less generalized backward bulging of the annulus. The nerve lies just anterior and in close proximity to the articular facet arising from the vertebra below. This is invariably the position of the root as it crosses the intervertebral disk. Any protrusion from or backward bulging of the disk margin tends to squeeze the root backward against the facet or against the ligamentum flavum.

The one outstanding symptom in these cases is pain — not a mild, sciatic pain but a severe, in-

tractable, disabling pain, which may have long remissions. Typically the pain runs down the back of the thigh and the outer side of the calf. Pain in the back may precede the leg pain, and a definite history of injury is given in about half the cases.

The one outstanding sign on physical examination is interference with straight leg-raising. This has been present in all our cases. Diminished or absent Achilles jerk occurs in 70 per cent, as does

TABLE 2. *Incidence of Traumatic Etiology, Symptoms and Physical Signs in Protrusion of the Fourth or Fifth Lumbar Disk (123 Cases).*

ETIOLOGY, SIGNS OR SYMPTOMS	INCIDENCE %	%
Trauma		49
From lifting	22	
From other causes	27	
Leg pain		100
Thigh only	13	
Thigh and calf	66	
Thigh, calf and foot	21	
Back pain preceded by leg pain		48
Stiff lumbar spine		70
Loss of lumbar curve		47
List or scoliosis		37
Diminished straight leg-raising		100
Diminished or absent ankle jerk		70
Sensory change (subjective or objective)		33

splinting of the lumbar spine. Loss of the lumbar curve, scoliosis and minor sensory changes are not uncommon. In severe cases motor weakness may be observed. Where the ruptured fragment is a large one, there may be evidence of compression of all the elements of the cauda equina with paralysis of the sphincters and so forth. This, however, is rare.

The total protein in the spinal fluid is usually somewhat increased (Fig. 2). Many cases show a level of 40 to 50 mg. per 100 cc., but in a very appreciable number the total protein, even in the first few drops drawn at the fifth lumbar space, is under 35 mg. Aside from this, the spinal fluid is generally normal.

We believe that the increased total protein in these cases is evidence of swelling and edema of the affected nerve roots above the point of compression. This condition has been noted many times at operation and can be demonstrated at times by examination with x-rays following Lipiodol injection. It may be that the serum protein in the swollen root passes through the arachnoid into the spinal fluid, thus raising the total protein.

The use of a contrast medium for x-ray examination of the spinal canal has been of inestimable value in the study of our cases, and in most of them we have employed Lipiodol (Fig. 3). This opaque oil gives an excellent contrast, lies in a thin layer against the anterior dura, and can be moved about readily with the patient prone on the tip table. According to Hampton,[10] it is by far the most satisfactory medium from the radiologist's point of view. We have long suspected that the occasional patient might give negative findings even with Lipiodol, and this has proved to be so in 2 cases. These patients were operated on notwithstanding a negative examination, and in each a ruptured fragment far to the side was found and removed.

The use of Lipiodol has certain disadvantages. It is so slowly absorbed that it may be considered a permanent foreign body. It is sufficiently irritating to cause considerable pain in the occa-

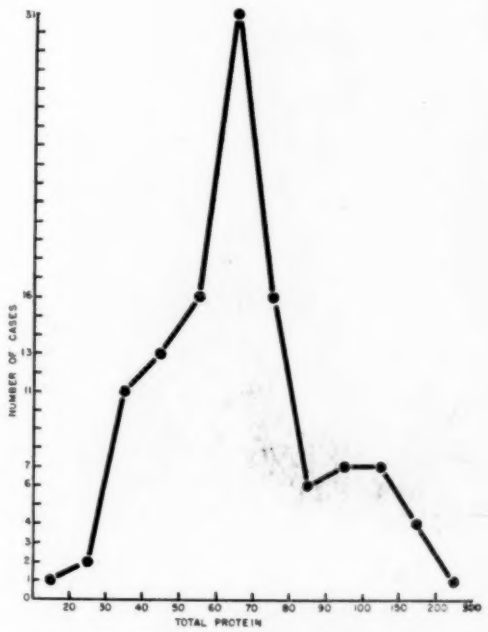
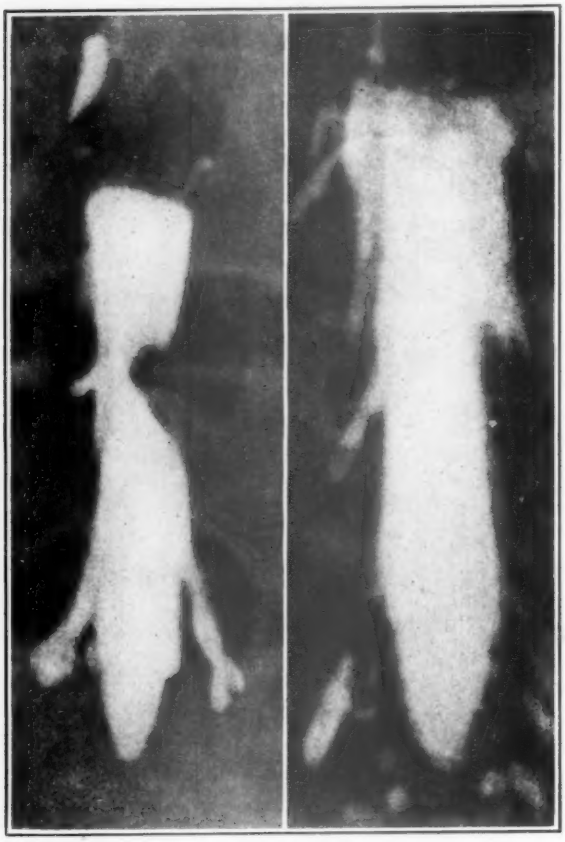


FIGURE 2. *Total Protein in the Spinal Fluid in Cases of Protruded Lumbar Intervertebral Disk.*

sional case. If the patient is tipped head down, either by accident or design, the Lipiodol may enter the cranium and become fixed there, occasionally causing severe intermittent headache. Lipiodol, therefore, is not used unless operative interference seems practically certain. In this we are in agreement with Love[11] and Camp,[12] who have handled a very large number of these cases. The Lipiodol is removed at operation, but this necessitates opening the dura, which would otherwise be unnecessary except in unusual cases.

The injection of air into the spinal canal gives satisfactory results in some cases (Fig. 4). We believe that Lipiodol will demonstrate a lesion in

some cases that would fail to be shown at all by injection of air. The contrast with air is poor, and the whole lumbar sac is filled, masking the defect, particularly if it be a small one. In some cases injection of air is used first. If it gives positive evidence of deformity of the lumbar sac, operation is indicated; if, however, the examination is negative or unsatisfactory, it is checked by examination with Lipiodol a week later. We have not used Thorotrast, as we have believed that it is too dangerous. It may be that we are in error in this regard. It is to be hoped that some new contrast medium will be found which will be as satisfactory as Lipiodol, but which will be absorbed readily and will have no ill effects.



A B

FIGURE 3. *Defects as Shown by Lipiodol.*
In A *there is a characteristic large defect; in* B, *a small filling defect of the right fifth-lumbar-root sheath.*

Spurling and Bradford[9] suggest that typical cases may be operated on without any investigation with a contrast medium. This is possible in some cases, but they must be very carefully chosen. Under these circumstances it may be necessary to explore both the fourth and the fifth disks on account of the similarity of the clinical pictures of each.

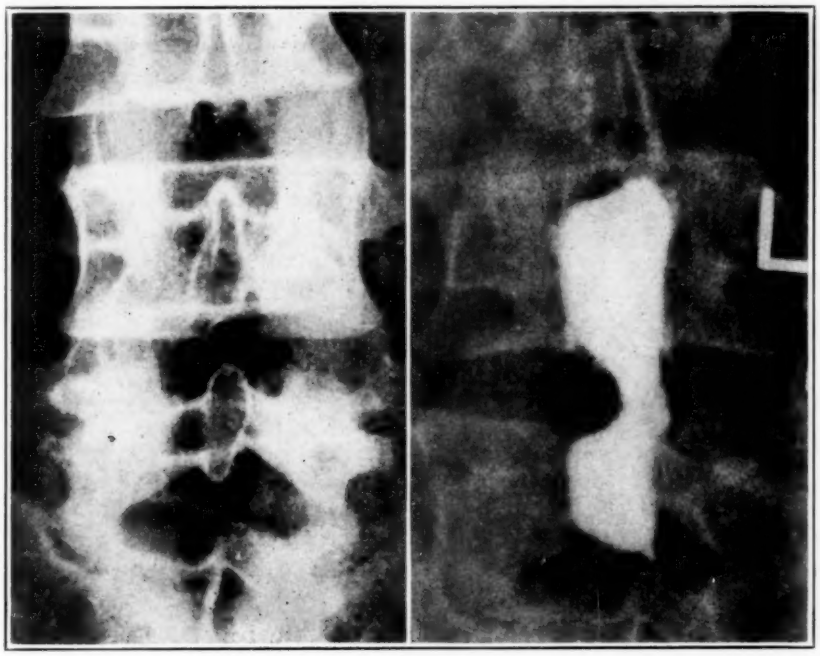
As already stated, rupture of the intervertebral disk is almost always at the fourth or fifth lumbar vertebra, and the symptom complex is constant. Notwithstanding this fact, a presumptive diagnosis of ruptured disk is often made and injection of Lipiodol carried out when such a procedure is not warranted. The promiscuous use of Lipiodol for patients suffering from low-back pain without the other signs enumerated above is dangerous and useless. The same is true of patients who have had symptoms for only a short time. The great majority recover spontaneously or under conservative orthopedic treatment. Investigation of the spinal canal for a ruptured disk should be reserved for those patients having severe, intractable sciatic pain accompanied by symptoms and signs strongly suggestive of this lesion. If this rule is followed, there will be fewer patients going about with Lipiodol in the lumbar sac or cranium.

Treatment of protruded disk may be operative, with removal of the fragment or stabilization of the spine by fusion, or may consist in non-operative measures such as rest in bed, or the use of a plaster jacket or some other form of corrective apparatus to hold the lumbar spine in flexion.

We realize fully that conservative measures or fusion will effect a symptomatic cure or temporary relief of symptoms in a certain number of these cases. We have no way of determining how many cases will be relieved or how many will recur, but we are of the opinion that the percentage of the latter will be large. Either of these forms of treatment requires a long period of immobilization. Operative removal seems far more logical, as the cause is definitely eliminated, the mortality is very low, and the period of disability is shorter than in the other methods of treatment.

Operative exposure of a protrusion may be carried out without the complete sacrifice of any lamina (Fig. 5). It is necessary only to cut away the lower edge of the lamina above and its upper edge below, as advised by Fincher and Walker.[13] Adequate exposure of the laminae is essential. The ligamentum flavum is resected and the laminae cut away, as stated above. If the lesion seems to be laterally placed, the inner edge of the articular facet arising from the vertebra below is trimmed off, the spinal canal is gently explored with the finger, and the lesion is demonstrated. If it is laterally situated, the dura and its contents are displaced mesially, the compressed root displaced and the mass is exposed.

The posterior longitudinal ligament generally covers the mass, and it is necessary to split it before the disk cartilage is visible. In some cases the ligament has already been split, and the projecting mass lies in direct contact with the dura and root sheaths. The mass is lifted out if free, or cut away if attached. An opening can usually be demonstrated, and an instrument passed into the nucleus pulposus. Bleeding from the plexus of veins lying anterior to the root sheath may be brisk, and is controlled by cotton or muscle packs. If the lesion seems to be mesial, transdural resection is carried out. The Lipiodol should be removed in every case.



A B

FIGURE 4. *Air Injection* (A) *and Lipiodol Injection* (B) *in the Same Patient.*

Note the sharp defect in the Lipiodol plate in contrast to the indistinct long defect in the air plate.

The aftercare of these patients is simple except where fusion has been combined with operative removal. Shells are not used. A simple dressing is all that is necessary, and the patient is moved freely. The wound is no more painful than is a laparotomy for gallstones, and the leg pain is generally relieved within a few days if not immediately. The patient is kept in bed about eighteen days and is then allowed to get up. A belt or brace may be used. Exercises to strengthen the back muscles are begun around this time, and the patient goes home about twenty-one days after operation. Lifting, driving an automobile or doing anything that may require a sudden strain is avoided for three months from the date of operation, and during this time muscle-building exercises are continued. At the end of three months light work is permitted, and at the end of six, heavy work. In a few cases where the patient's work is particularly heavy, such as piano-moving, we have advised a change of occupation. A few patients have developed toe-drop following operation, but this condition has never been permanent.

In our series one or more free fragments have been the most frequent finding, and break in continuity of the annulus with protrusion of an attached fragment of cartilage the second, while posterior bulging of the disk margin without a break in the annulus has been rare.

In the whole series, lesions of the fourth disk were commoner than those of the fifth. Thickening of the ligamentum flavum was encountered in association with protruded disk in 10 per cent of the cases, arachnoiditis in 3 per cent, and both in 1 per cent. It seems clear that this association points to a common cause — trauma. Sometimes the trauma results in protruded disk, sometimes in a thickened ligament, sometimes in arachnoiditis,

sometimes in a combination of two or all three of these lesions. But, as previously stated, protruded disk is by far the commonest of all. It may be that congenital weakness or degeneration of the annulus plays some part in the causation of this lesion and that the protrusion takes place at a weak spot.

We[7, 14, 15] have reported our immediate results in previous papers, and further experience confirms our opinion that the operative risk is not great. Of 123 consecutive cases of rupture in the low lumbar region proved by operation, there

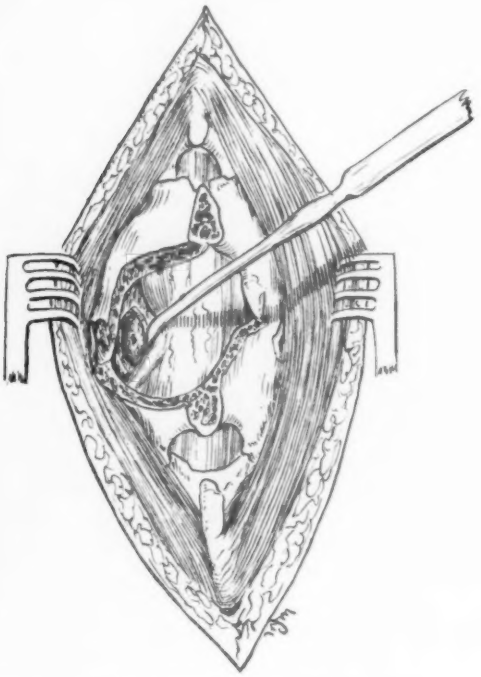


FIGURE 5. *Partial Laminectomy with Exposure of the Extruded Fragment.*

Note that neither lamina is cut across.

were 1 postoperative death and 1 case of later death from paraplegia, which was present preoperatively.

As regards the effect of operative removal of the extruded fragment, we have been able to trace 77 patients operated on over a year previously. We realize that one-year results may be too short to give accurate information. As reports of larger groups of cases with a longer elapsed period after operation are reported, some of our conclusions may have to be changed. The analysis of this group of cases is difficult, as the result must be measured largely by the personal estimate by the patient of the amount of his discomfort, and this estimate varies at different visits.

Thirty-six patients (47 per cent) are well and complain of no residual discomfort. Some of our patients classified as well are working at less arduous jobs than the ones they held before they were ill. Twenty-five (32 per cent) are effective in their work or in other activities but do not class themselves as absolutely well. Many of these complain that their backs are tired after a hard day or that they have a little discomfort in their legs at times. A few have slight limitation of straight leg-raising or diminished ankle jerk. They are all satisfied that they are greatly improved. Thus 80 per cent of the results are good.

Eight (10 per cent) of the results are fair. The patients are able to work but are handicapped by pain if they do too much. They are satisfied that they have been definitely improved by the operation. The poor results (10 per cent) are as follows. Three patients have improved but are unable to work. They have interference with straight leg-raising, stiff back, loss of ankle jerk and so forth. Five have severe low back pain or recurrence of leg pain with the signs of protruded disk. One of these cases was re-explored, and a definite recurrence of the protrusion was found.

We are under the impression that the patients who had fusions performed at the time of the laminectomy have shown slightly better results than those in whom no fusion was done. The difference is not great, and we can give a definite opinion on this point only after analysis of a larger group of cases than we have at present. Fusion following laminectomy or partial laminectomy adds to the length of the operation and to the operative risk, and convalescence, of course, is longer.

Although this series has been analyzed on the basis of one-year observations, a number of the patients have been followed much longer, with results fully as good as those in the more recent group. We believe that it is correct to say that the results of operation in this disease are good in 80 per cent, fair in 10 per cent and poor in 10 per cent.

SUMMARY

Protrusion of the fourth or fifth lumbar intervertebral disk is a distinct entity. The rupture usually occurs at the side of the canal, with compression of the fifth lumbar or first sacral nerve root. Other roots may be affected as well.

The symptom complex is quite constant and easily recognized. Sciatic pain, a stiff flat back, interference with straight leg-raising, absence of ankle jerk and elevation of the total protein in the spinal fluid are the most important features.

The diagnosis is confirmed by roentgenological

examination of the spinal canal following injection of a contrast medium.

The operative treatment is described, and the results obtained are given.

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ACUTE SURGICAL EMERGENCIES OF THE ABDOMEN IN PREGNANCY*

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ANY acute abdominal condition which occurs in the nonpregnant woman may occur in the pregnant woman, with the possible exception of hemorrhage from a ruptured graafian follicle. There are several emergencies which can occur only in pregnant women, for example, incarceration of the retroverted pregnant uterus, rupture of the uterus and ectopic pregnancy. Since we wish to consider only emergencies arising during the course of an intrauterine pregnancy, we have excluded ectopic pregnancies and traumatic ruptures of the uterus during delivery. We have also excluded traumatic lesions of the other abdominal organs.

Table 1 shows a list of acute abdominal conditions and the number of cases of each encountered at the Boston Lying-in Hospital from 1916 to 1938, inclusive. During this period there were 66,431 deliveries in and outside the hospital. The true incidence of these emergencies is undoubtedly higher than that shown in Table 1, because some cases were undoubtedly admitted to the surgical services of general hospitals.

There were no cases of acute pancreatitis. This condition has been reported as complicating pregnancy but it must be exceedingly rare, since it accounts for only about 1 per cent of all acute abdominal conditions, and furthermore is commoner in patients past forty. There were likewise no cases of perforation of an ulcer. Gastric or duodenal ulcer being relatively uncommon in women as compared with men, the probability of a pregnant woman's having a perforated ulcer must be very small.

Biliary colic and acute cholecystitis are not so uncommon during pregnancy, but only rarely do they require immediate surgical intervention.

TABLE 1. *Acute Abdominal Conditions in 66,431 Deliveries (1916–1938).*

CONDITION	NO. OF CASES
Acute pancreatitis	0
Perforated ulcer	0
Acute gall-bladder disease	3
Intestinal obstruction	1
Incarcerated hernia	1
Acute appendicitis	24
Ovarian cyst	10
Spontaneous rupture of uterus	14
Fibroid, with acute degeneration or twisted pedicle	7
Incarcerated retroverted uterus	1
Total	61

Twenty-three of our patients had symptoms of gall-bladder disease, but in only 3 were they severe or persistent enough to make operation necessary. Pregnancy does not materially interfere with either the diagnosis or the treatment of acute gall-bladder disease.

The single case of intestinal obstruction occurred in the seventh month of pregnancy. The diagnosis was not made early. The patient had been vomiting for two days when admitted, was in poor condition, and died immediately after operation under local anesthesia. The immediate cause of death was asphyxia from aspiration of vomitus. Operation revealed an obstruction of the

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small bowel due to postoperative adhesions. This is the type of obstruction most likely to occur in pregnant women. In both nonpregnant and pregnant women the signs and symptoms of acute obstruction are the same, and there is the same urgent necessity of early diagnosis and operation. The earliest symptom of acute small-bowel obstruction is pain, usually severe and referred to the epigastric or umbilical regions. It often comes in spasms but may be continuous, particularly if the bowel is strangulated. Vomiting is an almost constant symptom, but its time of onset varies, so that it may not be an early one. Tenderness and distention are not early symptoms. Obstipation is inevitable, but the first enema may produce a good fecal result and it may be hours before the fact of obstipation can be established.

Hernia is a rare cause of acute abdominal emergencies in pregnancy. In this series there were 26 cases with ventral or umbilical hernias, none of which caused acute symptoms. There were 3 cases with inguinal hernias, one of which was incarcerated and required operation.

Acute appendicitis is by far the commonest abdominal emergency occurring in pregnant women. In this series the diagnosis of acute appendicitis was made in 24 cases, giving an approximate incidence of 1 in 2800 deliveries. In 3 cases the condition subsided spontaneously and the patients were not operated on. They all went to term without further attacks. One patient had an interval appendectomy two weeks after a subacute attack, with an uneventful recovery. Sixteen of the remaining 20 patients were operated on before rupture of the appendix had occurred, and all recovered. The question of the influence of drainage of the peritoneal cavity in causing abortion or premature labor is of interest, but it must be remembered that we are dealing with small groups of cases, and that the picture might be quite different if large numbers were available. In 11 of our 16 cases no drainage was used. Four of these 11 patients miscarried or delivered prematurely, while this occurred in 4 out of 5 of the unruptured but drained cases.

There remain 4 cases in which the appendix had ruptured. Three of these patients died and the fourth recovered after a long, stormy convalescence. Two cases were referred to the hospital by physicians in the seventh month of pregnancy, after several days of illness. Both patients showed at operation a ruptured appendix and general peritonitis. Both went into labor and both died. One patient, four months pregnant, was admitted after several days of mild pain in the right lower quadrant of the abdomen and fever. These symptoms were not given the attention they deserved because on admission the patient was aborting. When, eight hours after the abortion was complete, the patient developed severe shock and upper abdominal pain, the diagnosis seemed very obscure. Forty-eight hours after admission, exploration revealed a ruptured appendix and general peritonitis. This patient had a long convalescence, complicated by a subdiaphragmatic abscess, but recovered. The fourth patient, who had severe cardiac disease, during the eighth month of her pregnancy was admitted with a history of epigastric pain, vomiting and diarrhea of two days' duration. The cardiac condition, plus the fact that labor started shortly after admission, diverted attention from the symptoms of appendicitis. She was delivered by cesarean section and died of general peritonitis on the eighth day. Autopsy revealed the source of the peritonitis as an abscess from a ruptured appendix.

Success in the treatment of acute appendicitis in the pregnant patient, as in the nonpregnant, depends on early diagnosis and operation before rupture of the appendix. The earliest symptom is pain, and typically it is first epigastric, shifting later to the right lower quadrant, but, as in the nonpregnant patient, this pattern may vary. The pain is not necessarily severe, and may be tolerated for some time before medical advice is sought.

Localized tenderness over the region of the appendix is probably the most consistent sign of appendicitis, and must never be ignored. In early pregnancy the appendix occupies its normal position and may, of course, extend down over the pelvic brim, so that a rectal examination should always be made. As the uterus increases in size, the cecum is forced upward and carries the appendix with it. Account of this must be taken in evaluating abdominal tenderness in pregnancy. Vomiting may be delayed or absent and involuntary spasm may not appear, even though the appendix is at the point of rupturing. It must be remembered that a physiologic leukocytosis occurs in pregnancy, so that the white-cell count may be 12,000 or 13,000, or even, rarely, as high as 16,000. The existence of a pregnancy should not deter one from an early operation, and needless to say, the pregnancy should not be interfered with.

Of the lesions which do not involve the uterus itself, ovarian cyst is second to appendicitis as a cause of acute abdominal emergencies. There were 10 such cases in this series, all of which were operated on successfully, except that 1 patient miscarried five days after operation. The diagnosis of ovarian cyst with twisted pedicle is generally easy, because the cyst can usually be felt if the

patient is examined carefully, under anesthesia if necessary. A cyst may be confused with a pedunculated fibroid, but if there are symptoms of strangulation the mistake is of no great practical importance. Sometimes in a well-advanced pregnancy a small cyst is situated so high that it cannot be reached on vaginal examination and yet is not felt by abdominal palpation. X-ray examinations may be of great help in such a case, since many of the cysts complicating pregnancy are dermoids. Of 25 cases in which the nature of the cyst was known, it was a dermoid in 12.

Of the lesions that involve the uterus itself, the commonest is spontaneous rupture. There were 14 cases in this series, 10 of which were those of patients with ruptures of cesarean scars. Of the other 4 cases, one was that of a patient with a bicornuate uterus, which ruptured during the fourth month, and the other ruptures occurred during prolonged labor. There was but 1 death, and that occurred in a case in which the rupture was not discovered. All the patients with rupture of a cesarean scar survived.

As a rule, when spontaneous rupture of the uterus occurs, the pain, the severe shock, the cessation of the fetal heart and sometimes the escape of the fetus into the abdominal cavity make the diagnosis easy. However, the pain may be very slight and may be obscured by labor pains, the bleeding may be negligible and shock absent, and the fetus may not escape through the rupture. Thus, it happens occasionally that when the abdomen is opened to perform a second section, a recent but entirely unsuspected rupture of the old scar is discovered. When such a symptomless rupture of the uterus occurs in a patient who is delivered vaginally, there is a grave abdominal emergency but little or no evidence of its existence. It may be diagnosed accidentally, as in a case (not included in this series) in which an extensive rupture of the lower segment just before normal delivery resulted in no change whatever in the patient's condition, and was discovered promptly only because the placenta escaped into the abdominal cavity; or such a rupture may be diagnosed only several days later when peritonitis has developed, or perhaps not until autopsy. Consequently, whenever a patient has been delivered vaginally who previously had a cesarean section, or whenever after vaginal delivery there is any reason whatsoever to suspect a rupture of the uterus, it is advisable to explore the uterus manually. In all cases of rupture of the uterus the treatment is immediate supravaginal hysterectomy.

Fibroids as a cause of acute abdominal emergencies during pregnancy ranked fourth in our series. There were 7 cases, in 4 of which the fibroid was pedunculated. In 5 cases myomectomy was done, and in 2, hysterectomy. All the patients recovered without complications, but 2 went into premature labor following myomectomy, 1 in the seventh month and 1 in the eighth. Fibroids are a fairly common complication of pregnancy and the diagnosis is usually obvious. In most cases they are of little or no clinical significance. They seldom cause acute symptoms. In this series there were 147 cases with fibroids, 16 of which showed pain and tenderness. In 9 cases these symptoms were transient, subsiding with two or three days' rest in bed; in the other 7 the pain and tenderness were more severe and persisted. Most of the patients showed a moderate leukocytosis, but only 3 had fever, and the highest temperature was 100°F. The occurrence of pain and tenderness in fibroids during pregnancy is not usually a genuine abdominal emergency. If the tumors are pedunculated, reasonably prompt removal is advisable, but if not it is better to wait. In many cases the symptoms subside and the patient goes to term or to viability, whereas from 30 to 40 per cent of patients subjected to myomectomy miscarry.

Retroversion of the pregnant uterus very rarely requires operation, and still more rarely does it produce the picture of an acute abdominal emergency. In the 1 case reported here the patient was four months pregnant and was starting to miscarry when first seen. The angulation of the cervix resulted in obstruction. Severe lower abdominal pain and tenderness, slight bleeding, a rapid pulse and a very tender pelvic mass led to a preoperative diagnosis of a strangulated cyst or a ruptured ectopic pregnancy. At operation the retroverted, adherent uterus was easily freed and brought forward. It was then emptied by hysterotomy, a procedure the advisability of which is debatable in such cases.

Summary

Any acute abdominal condition may occur in a pregnant woman that can occur in a nonpregnant one. The principles of recognizing and handling such conditions are the same in both groups. The prime necessity is not an immediate differential diagnosis but early recognition on the part of the general practitioner or obstetrician that an acute abdominal emergency may exist. He must maintain an attitude of suspicion with regard to abdominal pain. Persistent pain is the one early and almost invariable symptom of an acute abdominal emergency, and should receive immediate and thoughtful consideration. He must take care that the existence of a pregnancy does not in any way distract his attention from abdominal pain or lull his suspicions with regard to it.

264 Beacon Street

SEVERE HEMORRHAGE FROM THE HEAD AND NECK

Its Management, with Report of a Case Showing the Retroparotid-Space Syndrome

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BOSTON

SERIOUS flooding hemorrhage from the head, mouth or deep vessels of the neck may often demand quick resourcefulness to avert impending disaster. When the hemorrhage is not readily controlled by pressure, packing, hemostat or primary ligature, the course of events may be rapid and the outcome fatal. Rarely does one encounter a hemorrhage beyond immediate surgical control, except from advanced intraoral cancer with sudden erosion of the large vessels of the floor of the mouth and neck, or from unusually severe and deep lacerations of the neck, such as those caused by fragments of glass from a shattered automobile windshield or by a deeply penetrating stab or bullet wound.

The resources of the physician for controlling such a hemorrhage are considerable. They may often prove useful in the salvage of patients outside of cancer institutions, where such hemorrhages more commonly occur. In the patient with extensive cancer of the deeper recesses of the mouth, pharynx, larynx or neck, hemorrhage ranks high among the terminal events. It has been well recognized that intraoral cancer rarely spreads beyond metastatic areas in the neck. Remote metastases to other organs have been found only in isolated cases, and practically never have they been regarded as a chief cause of death. Fatality usually results from locally destructive effects, namely, massive necrosis of tissue and slough of the floor of the mouth and neck, ischemic degeneration and ulceration of the metastatic cervical nodes, with secondary streptococcal infection ending in terminal septicemia, hemorrhage, progressive inanition, terminal pneumonia or any combination of these. There is also the possibility of radiation necrosis in the regions of the large blood vessels. One may be confronted with any of these complications in managing a patient at home, particularly during intervals following radiation treatment.

More often in cancer institutions does one become expectant of, and experienced in, meeting sudden massive hemorrhage from the head, neck or mouth. External hemorrhage from the head or neck may be directly controlled by the use of hemostat, suture ligature or pressure by moist gauze packing, with supplementary proximal ligation of the major vessels if required. Bleeding from the mouth, pharynx or larynx, however, is not so directly controlled. It calls for effective indirect emergency measures, which are discussed herewith.

When a sudden, unheralded and massive effusion of blood appears at the mouth the difficulties

FIGURE 1. *The First Stage in Attempting to Control Sudden Severe Hemorrhage from the Mouth and Pharynx: Intraoral and extraoral digital pressure.*

are extreme, and unless a physician happens to be at hand, death soon occurs. Not infrequently, however, such premonitory signs as a previous, less severe hemorrhage or a slow trickling ooze may give warning of the likelihood of more serious, sudden hemorrhage. This allows the physician opportunity to consider the advisability of early proximal ligation of the affected vessel. This usually means ligation of either the external or, less often the internal, carotid artery, or control

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of both by ligation of the common carotid artery. In any event, further preparation for an emergency should be made by having at the bedside a set for emergency tracheotomy, a wooden gag to insert between the teeth to prevent biting, sterile albolene, mineral oil or olive oil, a 2-inch-wide gauze strip for tight packing of the oropharynx and a small suction apparatus for aspiration of obstructing blood from the trachea and bronchial airways.

When one is suddenly confronted with bleeding from the mouth, temporary control may be attempted, first by insertion of a wooden gag between the teeth. The index finger can then be safely inserted deep along the lateral wall of the pharynx, firm to hard counterpressure being applied at the same time over the carotid vessels in the neck with the thumb or with the fingers of the opposite hand (Fig. 1). Such digital pressure may be shifted quickly to the opposite wall, and in certain cases this helps to indicate the source of the hemorrhage, and to allow time for the orderly execution of measures for more permanent control.

This temporary procedure suffices only with moderate hemorrhage, and in such a case it may be worth while to maintain such pressure for a prolonged period in an attempt to induce spontaneous clotting. In the presence of more active bleeding, however, it is quickly realized that this is but the first step. In most cases immediate tracheotomy at the bedside must be carried out. Following this, the firm packing of the pharynx and mouth with a long, single strip of gauze soaked with albolene or mineral oil lends the greatest assurance for immediate control of the hemorrhage (Fig. 2). Gentle and repeated aspiration of blood from the tracheobronchial tree should next be carried out. The secretions can be kept fluid by the use of small amounts of sodium perborate in a 3 per cent solution, dropped directly into the tracheotomy tube, thus lessening the necessity for repeated aspiration of thickened secretions.* Arrangements can then be made, first for supporting the patient by transfusion and parenteral fluids, and next for early ligation of the eroded vessel or its proximal source as soon as the patient's condition permits. The tight gauze packing should be loosened within twenty-four hours, and should be removed from the oropharynx as early as possible in order to prevent extensive pressure necrosis of the pharyngeal wall, which is so prone to occur. Early trial removal is certainly advisable, with repacking of the pharynx only if desperately urgent. The latter measures are so radical and extreme that they are, of course, reserved for only the most serious hemorrhages defying lesser methods of control. They may overcome the acute emergency, and in an occasional case may be of considerable therapeutic effectiveness.

Deep lacerations and penetrating stab wounds of the head and neck largely arise from such instruments as broken glass, knives, bullets, shell fragments and other piercing metal objects. Wide tangential lacerations usually sever the relatively superficial anterior and lateral jugular veins, and occasionally the deeper-lying internal jugular vein. Penetrating stab wounds more often injure the internal jugular vein, the carotid arteries and the large tributaries of the external carotid artery. In most cases, however, the hemorrhage can be readily and directly controlled by adequate surgical exposure and accurate securing of the bleeding points with hemostat and ligature.

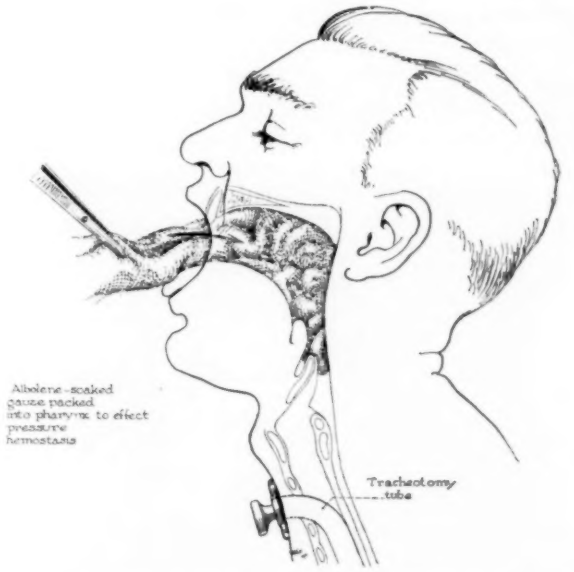

FIGURE 2. *The Second Stage in Attempting to Control Sudden Severe Hemorrhage from the Mouth and Pharynx: Emergency tracheotomy, tight packing of the pharynx with oil-soaked gauze, later ligation of the affected vessel in the neck and early removal of the pharyngeal pack to avoid pressure necrosis.*

Stab wounds at the level of the base of the skull (second and third cervical vertebrae) may give rise to hemorrhage especially difficult to control. Here the problem may become alarmingly acute and bring into play all available measures, both direct and indirect. Forceful digital pressure above and below the edges of the wound will usually allow time to get the patient to the hospital and to the operating room. Despite generous operative

*This method, employed on the Tracheotomy Service at the Haynes Memorial Hospital, was suggested by Dr. Conrad Wesselhoeft.

enlargement of the stab wound, both the depth of the bleeding area and the massive effusion of blood may frustrate a radical attempt to gain direct exposure of the bleeding vessel or vessels. In addition, the dire possibility of intravenous aspiration of air, leading to air embolus, must be faced. Shock and infection loom as further complicating factors.

Damage to the last four cranial nerves (the ninth, tenth, eleventh and twelfth) and to the cervical sympathetic trunk may occur from a knife or bullet wound through the retroparotid space, producing the serious and characteristic sequelae described by Villaret[4, 5] as the syndrome of the retroparotid space. Immediately after their exit from the skull, these four cranial nerves lie so close together as to simulate a plexus with the cervical sympathetic trunk. The hypoglossal nerve lies medial to the spinal accessory nerve and winds around the cervical sympathetic, the glossopharyngeal and the vagus nerves, from behind, forward and downward. Hence, injury by

TABLE 1. *Possible Results of Nerve Injury in the Retroparotid Space.*

RESULTS	NERVE INVOLVED
Loss of taste on posterior third of tongue Deviation of dorsal wall of pharynx to sound side Loss of sensation in pharynx, palate and fauces Loss of sensation on dorsal wall of external auditory meatus of ear	Glossopharyngeal (ninth cranial) nerve
Paralysis of vocal cord and palate on side of injury, with hoarseness and dysphonia Difficulty in swallowing and eating, with regurgitation of fluids into nasopharynx	Superior laryngeal or inferior recurrent laryngeal (vagus-tenth cranial) nerve
Inability to raise arm due to paralysis of trapezius muscle, causing a winged scapula deformity	Spinal accessory (eleventh cranial) nerve
Paralysis and atrophy of one side of tongue, with deviation to the injured side	Hypoglossal (twelfth cranial) nerve
Enophthalmos, narrowing of palpebral fissure, miosis and numbness of side of face—Horner's syndrome on side of injury	Cervical sympathetic nerve trunk

a stab wound or bullet in this area may involve any or all these nerves—Galen considered the ninth, tenth and eleventh cranial nerves as one, because they are so close together. After the publishing of Villaret's description, additional cases were reported by Pollock[2] in 1920 and Stookey[3] in 1922. The clinical findings resulting from such injury are characteristic, and may include some or all the changes listed below. Their recognition may prove helpful in locating the position of a bullet or shell fragment or the exact site of injury from a stab wound. Interestingly enough, the case reported here showed all the changes listed in Table 1.

While Villaret, as stated, regarded the syndrome of the retroparotid space as resulting from injury to the four cranial nerves and the cervical sympathetic trunk, Vernet (cited by Stookey[3]) described injuries of lesser extent, namely, to the ninth, tenth and eleventh cranial nerves, as the "syndrome of the foramen lacerum."

Injury to the phrenic nerve and to the trachea may further complicate the picture. Control of the hemorrhage remains the primary consideration, however, and this will pave the way for proper management of the accompanying injuries.

CASE REPORT

E. C., a 32-year-old shoe worker, was stabbed severely in the upper right posterior side of the neck with a 4-inch-long, flat-bladed shoe knife, on March 30, 1939. A fellow worker quickly applied manual pressure to stem the rapid flow of blood until the patient's physician, Dr. William A. Flynn, of Amesbury, Massachusetts, arrived five minutes later. At this time blood was pouring freely from the wound and the patient was coughing up blood from the nose and mouth. He was taken by ambulance to the Amesbury Hospital, where Dr. Flynn tightly placed three deep silk sutures through the skin and subcutaneous tissues, in order to close the superficial portions of the wound without drainage. Following this a pressure bandage was applied and parenteral fluids were administered, and the patient did reasonably well, considering the severe loss of blood, until the following morning. While the dressing was being changed he suddenly coughed and blood spurted forcefully from between the sutures. Further serious loss of blood was prevented by having a nurse apply firm digital pressure above and below the edges of the wound until arrangements for operative control could be carried out.

I was asked to see the patient 18 hours after admission to the hospital. Examination revealed a muscular, pale, weakened, conscious man who showed the effects of acute loss of blood. The pulse was 110. The blood pressure was 110/60. Just below the right mastoid process, in the upper right posterior side of the neck, there was an obliquely horizontal, sutured wound 4 cm. in length, through the apposed edges of which a large column of blood spurted whenever digital pressure was released. The red-cell count was 3,820,000, the hemoglobin 68 per cent (Sahli) and the white-cell count 14,950.

A recent attempt to examine the mouth had precipitated further moderate bleeding from the nose and mouth. Later examination confirmed my suspicion that the posterior pharyngeal wall had been pierced just above the level of the soft palate. The uvula and soft palate were grossly discolored, purple and swollen. There was paralysis of the right vocal cord, and a low husky pitch to the voice, with thickened speech, difficulty in swallowing and nasal regurgitation. The patient complained of constant accumulation of mucus in the throat.

Immediate operation was carried out under novocain anesthesia. The three widely spaced silk sutures were removed, when a stream of blood 2.5 cm. in diameter spouted forth from the extreme depth of the wound, to rise for 10 cm. above the level of the skin. This flow was controlled only by having the assistant maintain constant digital pressure along each edge of the wound. The deep clot was removed, the incision generously enlarged and the deep fascial layer firmly retracted with grasping clamps in a radical attempt to gain direct exposure of the bleeding point. Exploration revealed the depth of the wound to be more than 10 cm., the direction obliquely inward and slightly upward toward the pharynx and the

deep-tissue injury extensive, undoubtedly reaching the pharynx. After a reasonable attempt to control this terrific bleeding, which defied exposure, the wound was tightly packed with gauze soaked in sterile olive oil.

The patient was then turned on his left side, and under novocain infiltration a 5-cm. incision was made along the anterior border of the right sternomastoid muscle in the upper third of the neck. The muscle was retracted laterally, and the fibers of the underlying sternohyoid muscle were separated longitudinally to expose clearly the bifurcation of the common carotid artery. The external carotid artery was doubly ligated with No. 1 chromic

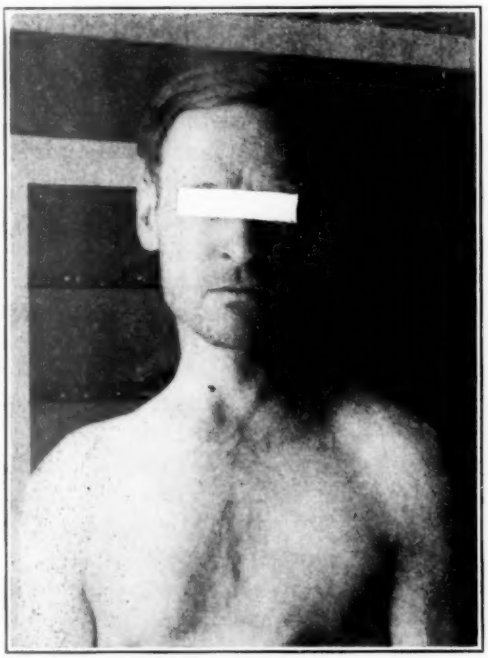

FIGURE 3. *Marked Atrophy of the Trapezius Muscle in the Right Neck and Shoulder Drop (photograph taken eleven months after injury).*

catgut above and below its first branch, the superior thyroid artery, thereby cutting off the possibility of collateral blood flow from the opposite side of the neck. The wound was closed in layers.

Following this procedure, attention was again turned to the stab wound in the neck. The pack was gently removed, and for 30 seconds nothing happened. Then blood again poured forth from the depth of the wound, in an equally large stream but with less force. It appeared to be coming from the internal jugular vein at the base of the skull, with the bleeding from the posterior auricular and occipital arteries probably removed. The wound was tightly repacked with oil-soaked gauze, a tight pressure bandage was applied, and the patient was transfused. Shortly thereafter he was transferred to the Massachusetts Memorial Hospitals, Boston, both for measures of direct control on later removal of the pack, and for the facilities of a blood bank, if needed.

Forty-eight hours later, on April 1, half the pack was removed and in 72 hours the entire pack was removed. Each time the wound immediately filled with slowly welling blood, especially on complete removal of the pack. A thin gauze strip covered with vaseline was lightly inserted, and the bleeding subsided within the next 5 hours. The patient's course henceforth was good, and it was evident that the emergency had passed.

In order to correct the large, gaping wound cavity, a secondary closure was carried out on the 8th day after the accident. It is of interest that the patient complained at this time of fairly severe and almost constant right-sided headache, lasting since ligation of the external carotid artery, and that this persisted with slowly diminishing intensity throughout his 10 days' hospital stay. That the right vagus nerve had been simultaneously injured was evidenced by the paralysis of the right vocal cord (recurrent laryngeal nerve), the low, husky voice, the difficulty in swallowing and the constant irritation from the pooling of thick mucus and saliva in the throat (superior laryngeal nerve), of which the patient could not rid himself. He was discharged on April 8, the tenth day after the injury, with marked improvement in his general condition, but with little change in the local disturbances noted above. At that time the red-cell count had risen to 4,070,000, and the hemoglobin to 74 per cent.

Two weeks after discharge, both wounds were well healed and the patient's general condition was good. He

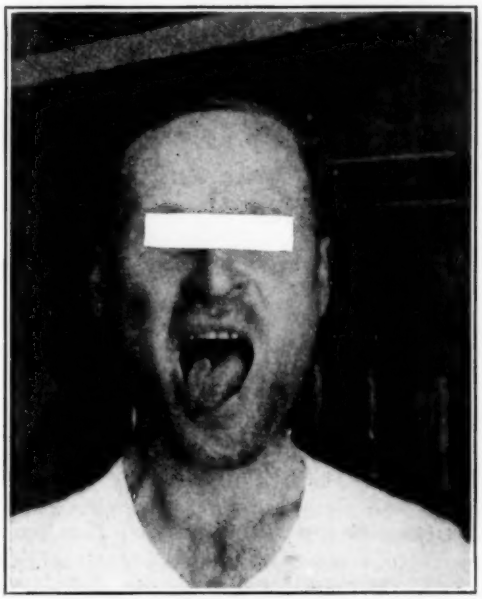

FIGURE 4. *Hemiatrophy of the Tongue and Marked Deviation to the Injured Side (photograph taken eleven months after injury).*

still complained of occasional headaches across the entire frontal region.

Seven weeks after the injury, on May 22, the patient stated that he was well except for much trouble with his throat; that is, it was hard to swallow, especially solid foods, which threatened to go down the trachea. He had much phlegm daily, with marked hoarseness and lowered pitch to the voice. Whereas he had had no headaches before operation, he had morning headaches almost every day, and occasionally during the day. In addition, he called attention to the difference in the size of his eyes. He had a definite right enophthalmos and dilatation of the pupil, and complained of marked numbness of the entire right side of the face, especially when shaving. Thus, a typical Horner's syndrome had resulted from the apparent

severance of the cervical sympathetic chain at the time of the original injury.

Eleven months after the injury, March 6, 1940, the patient halted his heavy outdoor work on the highway to appear for examination. By this time perusal of the literature had drawn my attention to Villaret's syndrome of the retroparotid space. Curiously enough, this patient showed all the evidences of complete extracranial division of the glossopharyngeal, vagus, spinal accessory and hypoglossal nerves, and partial recovery from injury to the cervical sympathetic nerves (Figs. 3 and 4). The syndrome was evident in its entirety.

Signs of Horner's syndrome, though still present, were lessened, in that the enophthalmos and lid narrowing were slight, the pupil was no longer dilated, and the numbness of the right side of the face had disappeared. To

FIGURE 5. *End Result.*

Both incisions are well healed, and the patient shows a greatly improved nutritional state. This photograph was taken eleven months after injury.

the remaining changes, which had become permanent, the patient had adjusted himself admirably. The voice was low and husky but tired less easily. The right vocal cord was immobile and in the cadaveric position. Mucus in the throat, difficulty in expelling it and dysphagia were still annoying. There was marked atrophy of the right half of the tongue, with sharp deviation to the affected side. There was loss of taste on the right posterior half of the tongue, and numbness of the right external auditory canal. Marked wing deformity of the scapula was present, with atrophy and paralysis of the right trapezius muscle and inability to raise the right arm above the level of the shoulder.

Additionally, the patient stated that 2 months after the injury, or 9 months previously, an abscess had drained for 1 week through the right auditory canal, and that complete deafness of the right ear had resulted. He also complained of steady, throbbing tinnitus of the right side of the head whereby he was conscious of every heartbeat. His headaches had ceased. Both wounds were well healed and the scars not conspicuous (Fig. 5). Despite the disturbances noted the patient was in excellent general health, enjoying life and being actively engaged in outdoor manual labor.

In this case, ligation of the external carotid artery diminished but did not in any manner control the hemorrhage. The persistent nonarterial bleeding suggested severance of the internal jugular vein at a level close to the base of the skull, just below its emergence from the jugular foramen. At the suggestion of Dr. Alexander S. Begg, of Boston University School of Medicine, evidence of this severance was sought by examining the head of a cross-sectioned cadaver at the level of injury. This allowed clearer visualization of the path of the flat-bladed knife through the deep retroparotid space to the nasopharynx (Fig. 6). It became apparent that the knife blade had

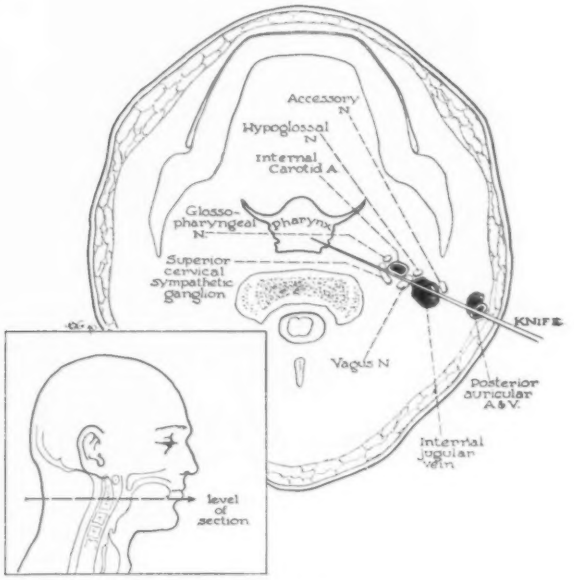

FIGURE 6.

This sketch shows the level of injury (insert) and a cross-section study. The path of the knife blade traverses the retroparotid space. Note the closeness of the uninjured internal carotid artery to the other structures severed, namely, the posterior auricular artery and vein, internal jugular vein, last four cranial nerves, and cervical sympathetic ganglion.

coursed medially, inward and slightly upward, to pass close to the body of the third cervical vertebra, and to sever the posterior auricular artery, the internal jugular vein, the right glossopharyngeal, vagus, spinal accessory and hypoglossal nerves and the cervical sympathetic chain at this high level, and finally to reach and pierce the nasopharynx. Examination of the cross-sectioned head clearly indicated that the knife could not have reached the pharynx along this course without having severed the internal jugular vein. Furthermore, the clinical evidence of injury to the right ninth, tenth, eleventh and twelfth cranial nerves and to the

cervical sympathetic trunk was substantiated by the close anatomic relation of these structures to the internal jugular vein.

Ligation of the external carotid artery without delay, in my opinion, was urgently necessary before placing reliance on gauze packing to control the persistent venous bleeding. It should be mentioned that while the danger of ligating this artery is practically nil if thrombosis extending to the internal carotid artery is avoided by ligating the artery distal to the superior thyroid artery, nevertheless permanent ligation should be done only when absolutely necessary. Neither the condition of the patient nor the circumstances for control of aftercare made temporary occlusion of the artery, by passing an untied ligature around it and strapping it to the skin under traction, dependable or safe.

Ligation distal to the superior thyroid artery is regarded as the site of election, in that it is technically easier, does not endanger the internal carotid artery with thrombosis and cerebral embolism and lessens the collateral blood supply from the opposite side by way of the superior thyroid artery. It must be stated that in the reported case the external carotid artery was ligated both above and below the superior thyroid artery. Fortunately no hazardous sequelae resulted, save for fairly persistent right-sided temporofrontal headache. This gives rise to speculation as to the possibility of avoiding these cerebral symptoms by ligating distally and not proximally to the superior thyroid artery.

Summary

With passing mention of the usual systematic procedures widely employed in the direct control of hemorrhage, emphasis has been more particularly directed toward the management of sudden, severe hemorrhage arising, from the mouth, as in the cancer patient, or from the head and neck following especially deep lacerations of the stab-wound type.

Suggestions for handling such an emergency have been mentioned in detail, with report of a patient manifesting sudden severe hemorrhage from the neck and mouth and extensive injuries to the extracranial nerves. Follow-up examination eleven months after the injury showed all the changes suggested by Villaret in his description of the syndrome of the retroparotid space. The case report is presented as substantiating the accuracy of Villaret's original description of this characteristic post-traumatic nerve-injury syndrome.

The site of election for ligation of the external carotid artery is emphasized for the purpose of preventing any likelihood of cerebral thrombosis and embolism, and of effectively eliminating collateral blood flow from the opposite side through the superior thyroid artery.

Conclusions

In the majority of even severe hemorrhages, adequate control may be gained by the use of hemostat, ligature, pressure or packing.

Occasionally added resources may become urgently necessary in order to control hemorrhage from intraoral cancer and deep lacerations of the head and neck, especially of the stab- and bullet-wound types.

Although in many cases massive hemorrhage from the mouth may rapidly become fatal, methods of quick, orderly approach such as immediate intraoral and extraoral manual pressure, tracheotomy, followed by gauze packing of the pharynx, and later proximal ligation of the affected vessel, may help in controlling the emergency and in the eventual salvage of the patient.

Likewise, deep lacerations of the stab- and bullet-wound types may require any or all these measures to control the hemorrhage.

Injury in the region of the retroparotid space is very likely to sever the last four cranial nerves — glossopharyngeal, vagus, spinal accessory and hypoglossal — and the cervical sympathetic trunk. This gives rise to characteristic changes described by Villaret in 1917 as the syndrome of the retroparotid space.

The characteristics of this syndrome are easily recognizable, seriously damaging and usually permanent.

Control of such massive hemorrhage and recognition of these extracranial-nerve injuries have been illustrated in the case reported here. Villaret's syndrome was evident in its entirety, especially at examination eleven months after injury.

Ligation of the external carotid artery should be done only when urgently necessary, for complications do occur. The artery in most cases should be ligated just distally to its first branch, the superior thyroid artery, in order to prevent thrombosis extending to the internal carotid artery, and to eliminate collateral blood flow from the opposite side via the superior thyroid artery.

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THE CLINICAL MANAGEMENT OF BREAST TUMORS*

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THERE are two questions of practical importance in the clinical management of breast tumors: What is the nature of the tumor? What is the appropriate treatment for this type of tumor? It is at once obvious that the answer to the second question cannot be arrived at until the first has been answered completely and accurately. It has seemed worth while to inquire into the accuracy of the clinical diagnosis of breast tumors. A review has therefore been made of the 127 breast tumors subjected to operation at the Pondville Hospital in 1938 and 1939.

Our policy is to recommend prompt exploration in all cases of breast tumor. The patient's permission to carry out whatever surgery may be necessary is secured. The exploration is usually performed under general anesthesia, and it is planned to have a pathologist present at the operation when there is any doubt about the diagnosis. A comparison was made between the preoperative and postoperative diagnoses in our series. There was a clinical error in diagnosis in 28 cases, or 22 per cent. This is a rather high percentage of error, but it does not begin to describe the difficulties in clinical diagnosis. In many cases a presumptive diagnosis which eventually proved to be accurate had been made, but it was necessary to confirm it by immediate pathological examination. It is interesting to find that in 45 cases the tissue was subjected to frozen-section diagnosis, and that in an additional 36 cases immediate gross pathological examination was carried out in order to confirm the diagnosis. Thus, in 56 per cent of the cases pathological help was employed at the time of operation (Table 1).

It may at once be pointed out that many of the operations at the Pondville Hospital are carried out by younger men who have less extensive experience with the clinical aspects of these diseases, and who derive some satisfaction from immediate pathological confirmation of the diagnosis. On this account it may be urged that the number of immediate pathological examinations is excessive. If in the hands of more experienced surgeons it is necessary in fewer cases to have a pathologist present at the time of operation, this means simply that in these cases the surgeon is acting as his own pathologist in interpreting the gross appearance of the tumor and in basing his decision on this finding. In this sense it may be said that the more experienced surgeons depend on pathological examination just as freely as do the younger men. Furthermore, a resident pathological staff is ready and willing at all times to be present in the operating room to carry out immediate examinations, and perhaps for this reason an excessive number of such examinations are made. In other hospitals and clinics it may be less convenient or feasible to have a trained pathologist present, but that does not mean that the patient's interests are being well served by the omission of such an examination. In 5 cases in our group of 45 frozen-section examinations the final pathological slides did not confirm the first diagnoses, so that even taking all possible precautions against errors, there is still a chance of inaccurate diagnosis.

TABLE 1. *Diagnosis of Breast Tumors.*

FINAL DIAGNOSIS	NO. OF CASES	ERRORS IN CLINICAL DIAGNOSES		IMMEDIATE PATHOLOGICAL EXAMINATION	
		NO.	PER CENT	NO.	PER CENT
Carcinoma	68	7	10	29	43
Chronic cystic mastitis	25	10	40	15	60
Adenofibroma	17	1	6	12	70
Papilloma	7	5	71	5	71
Miscellaneous	10	5	50	10	100
Totals	127	28		71	
General averages			22		56
Frozen-section diagnoses				45	
Errors in frozen-section diagnoses				5	

In endeavoring to answer the second question — What is the appropriate treatment for this type of tumor? — it may be desirable to ask an additional question in regard to malignant cases; namely, What is the extent of the disease? Taylor and Bruce[1] have pointed out elsewhere that actual clinical appraisal of the extent of the disease is subject to considerable inaccuracy. In the study of axillary examinations in cases of breast cancer we found an error of clinical appraisal in 28 per cent of the examinations. It is probable that errors as regards the extension to inaccessible parts of the body are even greater than this figure, in spite of careful x-ray examinations to detect remote metastases. It is sufficient to say that every care must be taken to make sure that the patient is in an operable stage before treatment is begun.

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When a patient is found to be in this stage and the diagnosis of cancer has been established, if necessary by immediate pathological examination, the treatment of choice is immediate radical breast amputation. This involves removal of the entire breast with its overlying skin, both pectoral muscles, and the axillary lymph nodes and fat as high as the clavicle. No alternative method of treatment can be shown to yield as consistently favorable results as can be secured by radical surgery.

The adenofibromas comprise a very interesting group of tumors which are benign in their behavior. There are no outstanding clinical characteristics aside from the presence of tumor that can be positively depended on. There were 17 cases, and frozen-section diagnosis was employed in over half of them. It is significant that an erroneous clinical diagnosis of adenofibroma was made in an additional 8 cases, 2 of which proved to be cancer. Excision of the tumor alone seems to be all that is necessary in cases of adenofibroma.

There were 25 operated cases in which chronic cystic mastitis was found. A correct clinical diagnosis was made preoperatively in only 15 of these. An erroneous clinical diagnosis of cystic mastitis was made in 7 cases, 3 of which proved to be carcinoma. Immediate pathological examination was employed in 15 of the cases that proved to be chronic cystic mastitis. The proper method of treatment for this condition has not been fully established. In our group, partial resection of the breast was carried out in 12 cases and mastectomy in 13. It has recently been pointed out that chronic cystic mastitis actually constitutes a precancerous condition, in that the breast that is the seat of the disease is more likely to develop cancer subsequently than is a normal breast.[2] However, this likelihood does not seem to be great enough to warrant advocating a bilateral simple mastectomy in all cases. In our series, operation was carried out in most cases in order to rule out the possibility of cancer.

Intraductal papilloma was found at operation in 7 cases, but a correct clinical diagnosis was made preoperatively in only 2 of these. Immediate pathological examination was carried out in 5 cases, and in 2 of these the frozen-section diagnosis suggested carcinoma so strongly that radical mastectomy was performed. In the remaining cases, excision of the tumor was carried out in 2 and simple mastectomy in 3. This condition also can reasonably be considered to be precancerous, and it is not uncommon to find multiple intraductal papillomas in these patients. Proper surgical procedure in this group also is not fully established. In these cases, as in those of chronic cystic mastitis, it is undoubtedly important to carry out protracted observation because of the possible later development of cancer.

There were 2 patients suffering from sarcoma of the breast, and in 1 of these the clinical diagnosis had not indicated malignancy. Both cases were treated by simple mastectomy. It is probably unnecessary to carry out a radical mastectomy in cases of sarcoma, although it is desirable to remove the fascia overlying the pectoralis muscle, or perhaps even to remove the muscle itself. The disease is slow to metastasize, and most failures in treatment are due to local recurrences. Thus it would seem desirable to be more radical in the treatment of the disease, especially in regard to the extent of dermal and fascial removal.

In 8 cases the final diagnosis proved to involve other conditions such as inflammation, localized area of hypertrophy and fat necrosis. Immediate pathological examination was carried out in all these cases, and all were subjected to operation on the ground that cancer could not be ruled out on clinical evidence.

Conclusions

It is evident that no breast tumor can be absolutely declared to be noncancerous on the basis of clinical examination alone. Its presence is presumptive evidence of cancer until pathological study has demonstrated the contrary. This means that every breast tumor should be subjected to exploration. The decision as to the proper method of treatment can be based only on accurate knowledge of the nature of the disease.

Benign conditions that are encountered at exploration are probably adequately treated by removal for examination. Malignant tumors should be subjected to radical surgery without undue delay. It is only by the following of these principles that any considerable improvement in the management of tumors of the breast can be expected.

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ROCKY MOUNTAIN SPOTTED FEVER*

An Analysis of Seven Cases, Including One Laboratory Infection

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RECENT investigations of the rickettsial diseases by Zinsser,[1] Dyer,[2] Lillie,[3] Topping[4] and others have added materially to our knowledge of this group of infections. Rocky Mountain spotted fever, first described by a United States Army surgeon[5] in 1896, was found by Wilson and Chowning[6] in 1904 to be transmitted by a tick. The etiologic agent was identified by Ricketts[7] in 1909. It is a truly American disease.

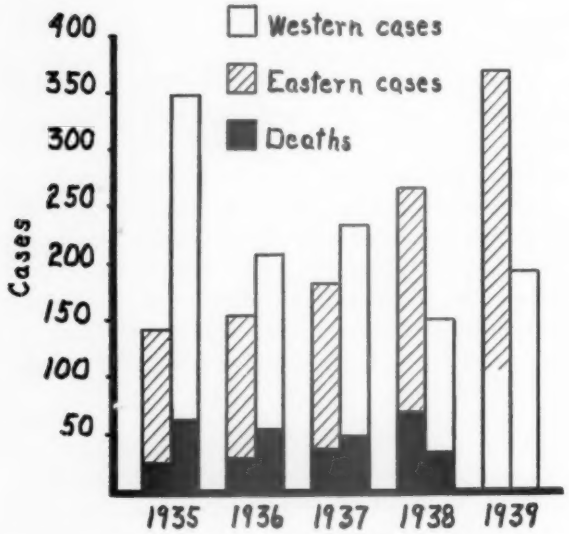

FIGURE 1. *Case and Death Incidences of Rocky Mountain Spotted Fever in the United States (1935–1939).*

The western cases include those from Montana, Idaho, Wyoming, Colorado, New Mexico, Arizona, Utah, Nevada, Washington, Oregon and California; the eastern cases, those from all the other states. No cases were reported from Maine, New Hampshire, Vermont, Connecticut and Wisconsin.

Much interest has been aroused by the increasing incidence of Rocky Mountain spotted fever in the eastern United States (Fig. 1). Only five of the forty-eight states have yet to report this disease, and although the incidence is not great, enough deaths have occurred to make it a serious medical problem. Aside from an academic interest in the relation between the Eastern and Western varieties, there is little if any clinical basis for differentiating them.

From 1931 to 1939, 7 patients with Rocky Mountain spotted fever were treated at the Walter Reed General Hospital in Washington. Three of them died. One patient, a physician, contracted the Western type of infection while working with the virus in the laboratory. A report of this case follows.

N. T., a 31-year-old physician, was admitted to the Walter Reed General Hospital on December 30, 1938, because of his own diagnosis of Rocky Mountain spotted fever.

The past and family histories were not important except that 2 months before onset the patient had received part of a course of Parker–Spencer vaccine, which caused his Weil–Felix reaction to be positive in a titer of 1:80. During the course of certain experimental studies on virulent Western tick virus he accidentally became infected, 11 days before admission and 6 days before the first symptoms of general malaise and headache. During the 5 days preceding hospitalization the patient noted pain in the eye muscles and joints, weakness, chills, fever and finally a rash on the hands.

Physical examination revealed a dejected man with a "hangover" appearance. There was a sparse petechial rash on the back of the hands. The pharynx was injected. The temperature was 103.8°F., the pulse 111, and the respirations 24. Otherwise the examination was normal.

The essential laboratory findings are summarized in Table 1 (Case 1). The Weil–Felix reaction, which was positive in a titer of 1:80 on admission, became negative following the infection and remained so for 1 year. Roentgenograms, electrocardiograms, blood cultures and other laboratory studies were normal. The Western type of virus was recovered from a guinea pig injected with the patient's blood on the 4th day after onset.

The febrile period lasted 19 days, the fastigium occurring on the 13th day with a temperature of 105°F. The temperature varied between 100 and 105°F., with the lowest levels occurring during the morning hours. The pulse varied from 90 to 140, and in general varied directly with the temperature. The rash spread to involve the whole body (Fig. 2), including the palms of the hands, the soles of the feet and even the optic fundi. It began to fade on the 20th day, giving place to a fine, powdery desquamation. Throughout the course of the disease the patient complained of severe photophobia, diplopia, soreness of the throat and pains in the muscles and joints. Marked mental cloudiness and irritability were the only neurologic features noted. He eventually made a complete recovery.

A study of the seasonal prevalence of Rocky Mountain spotted fever by Hampton and Eubank[8] shows that the season of high incidence for the Eastern variety begins in May, reaches a peak in

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July and declines in August and September. Of our 6 cases of the Eastern type, 1 occurred in May, 1 in June, 2 in July, 1 in August and 1 in November. Five cases were related to the bite of a tick. The sixth was an accidental laboratory infection with the Western tick virus. The seventh was contracted from an unknown source.

In 4 cases the period of time from the tick bite or the introduction of the virus to the onset of symptoms was given as five days in 1 case, six in 1 and seven in 2. In the fifth patient, a dog fancier, the incubation period could not be determined because he had been picking ticks from his dogs for several days. The sixth patient contracted the infection while hunting rabbits, seven days before his first symptoms. The seventh patient had taken several horseback rides into the mountains near Front Royal, Virginia, and had removed ticks from his body on many occasions. The appearance of the rash in 5 cases followed the onset of symptoms in one, two, three, three and five days, respectively.

It is unusual for a case of the Western type and one of the Eastern type to be treated in the same hospital at the same time. The courses of Cases 1 and 3 given in Table 1 overlapped, offering an opportunity to compare the clinical features of the two varieties. In Case 1, reported above, the physician-patient made many interesting and accurate observations of the symptoms and course of events. In comparison with the Eastern type of disease in Case 3, the Western type of infection was more severe: there was a more intense and widely distributed rash; the febrile period was longer, and the average elevation of the fever was well above that of Case 3. The headache, muscle pains and arthritic symptoms were more severe. Small petechial retinal hemorrhages were noted, but were not found in 2 of the patients infected with the Eastern type of virus. The prophylactic injection of the vaccine in Case 1 may have altered the features of the disease to some extent, but we believe that the characteristics serving to differentiate these cases were in no way sufficient in themselves to be of general differential value. The incidence of the various signs and symptoms occurring in the 7 cases is given in Table 2.

Case fatality studies of 2036 cases were made by Hampton and Eubank[8] in 1937. They found that during the period from 1933 to 1937, 1435 cases of the Western type had a fatality rate of 19.4 per cent. During the same period 601 cases of the Eastern type had a fatality rate of 18.1 per cent. There was thus little difference in the fatality in the two varieties. Our 6 cases of the Eastern type with a 50 per cent fatality constitute too small a sample to make possible more than a casual comparison.

It is customary to consider the possibility of measles, scarlet fever, chicken pox, typhoid fever, meningococcal meningitis, typhus fever, Rocky Mountain spotted fever and meningococcemia in cases with an unexplained fever associated with a rash. Despite this fact, it seems probable that many cases of mild infection are passed over too lightly by the busy physician and go unrecognized. It is important to encourage careful habits of thinking on the part of physicians in order that the chief characteristics of the case in question shall bring to mind a number of diagnostic possibilities.

TABLE 1. *Essential Laboratory Data* in 7 Cases of Rocky Mountain Spotted Fever.*

CASE NO.	AGE	SEX	WHITE-CELL COUNT $\times 10^3$	WEIL–FELIX REACTION	REMARKS
1	31	M	12.0–22.0	Positive, 1:80 (*Proteus* OX 2-19)	Recovered; guinea pig inoculated on 4th day of illness positive for Western type of virus.
2	41	M	12.5–14.5	Positive, 1:40 (*Proteus* OX 19c)	Died; necropsy.
3	42	M	6.0–19.0	Positive, 1:1280 (*Proteus* X)	Recovered
4	39	M	9.0–18.0	Positive, 1:160 (*Proteus* OX 2-19)	Recovered
5	21	M	6.0–21.0	Negative	Died; necropsy.
6	28	M	7.0–11.0	Positive, 1:640 (*Proteus* OX 19t-c)	Recovered
7	7	M	5.0–16.0	Positive, 1:5120 (*Proteus* OX 19)	Died; necropsy.†

*Albumin, casts and cells were commonly found in the urine from all cases during the febrile period.
†Necropsy findings previously reported.[15]

TABLE 2. *Signs and Symptoms Occurring in 7 Cases of Rocky Mountain Spotted Fever.*

SIGN OR SYMPTOM	NO. OF CASES	SIGN OR SYMPTOM	NO. OF CASES
Rash	7	Desquamation	2
Fever	7	Photophobia	2
Mental disorientation and irritability	6	Nausea	2
Headache	5	Weakness	2
Chills	5	Convulsions	1
Chemosis and injection of conjunctivae	5	Diarrhea	1
Generalized aches and pains	4	Jaundice	1
Meningeal irritation	3	Vomiting	1
Palpable spleen	3	Pains in muscles	1
Pneumonia	3	Sweating	1
Coated tongue	3	Anorexia	1
		Cough	1
		Hiccough	1
		Diplopia	1

The situation in which the patient is acutely and seriously ill with the cardinal symptoms noted above deserves prompt and thorough study. Time is often a chief consideration in the patient's chances for recovery. Once the proper diagnostic suggestion has been made, the progress of the case, in addition to the results of diagnostic procedures, seldom leads one away from the correct diagnosis.

FIGURE 2. *Fully Developed Rash in a Case of Western Type of Rocky Mountain Spotted Fever (United States Army Medical Museum, Negative No. 67987).*

The Weil-Felix reaction usually becomes positive between the eleventh and nineteenth days.[9] To ensure the best results three tests should be made: one as soon as possible after the onset, the second after ten days and the third during convalescence. The rise and fall of the agglutination titer is of great diagnostic importance. A titer of 1:160 in a case in which the first Weil-Felix reaction was negative is diagnostic. In some cases the titer may go as high as 1:5120. Depression of a vaccine-produced titer of 1:80 occurred in Case 1. Even after a year, no agglutinins for the various OX strains of the proteus bacillus had appeared in the blood. A satisfactory explanation for this curious though not unique phenomenon has not been advanced. By analogy it seems reasonable to suppose that the degree of immunity cannot be measured by the degree of agglutination titer.

In the Western type of infection in guinea pigs a scrotal reaction, consisting of edema, redness and occasionally necrosis, results from the rickettsial invasion of the skin and subcutaneous tissues. This type of reaction has not been observed in guinea pigs inoculated with the Eastern strains of the virus. The test was performed in 3 of our 7 cases; a positive result was secured in 1 (Case 1). Scrotal edema and necrosis in human beings was noted by Wood[5] in his series of collected reports published in 1896.

The significant necropsy findings in our 3 fatal cases were generalized petechial and hemorrhagic skin eruptions. Subserous hemorrhages were found in the heart, brain, kidneys, bladder and lungs. Areas of focal necrosis were noted in the heart, brain, spleen, kidneys and testes in Case 2. Acute splenic tumors — 200 gm. in Case 2, 380 gm. in Case 5 — of the septic type with sinusoidal engorgement were found in 2 cases. The third (Case 7) showed a spleen weighing 150 gm. with hemorrhagic foci and small pools of blood involving the Malpighian corpuscles. Bronchopneumonia was present in all 3 cases. Cortical hemorrhages occurred in the right adrenal gland in Case 5. Perivascular and petechial hemorrhages were found throughout the white matter of the brain in all cases.

Treatment consists of general measures to maintain the patient throughout the febrile period, such as the administration of intravenous fluids, sedatives, and digitalis if signs of myocardial failure or arrhythmia appear. There is no specific treatment. Vaccines, useful in prevention, are of no value. Sulfanilamide was tried in Case 3, without benefit. Others[10, 11] have reported un-

favorably on the use of both sulfanilamide and sulfapyridine. The production of a potent antiserum has been attempted by Ricketts and Gomez,[12] by Heinemann and Moore[13] and by Noguchi.[14] No record was made by any of these authors of the use of antiserum in human cases. Topping[4] reports the beneficial effects of immune serum in the treatment of this disease in guinea pigs and monkeys. It is hoped that his studies will lead to the development of a specific therapeutic agent with which to treat this highly fatal malady in man.

Summary

The clinical and laboratory features of 6 cases of the Eastern type of Rocky Mountain spotted fever, with necropsy findings in 3 cases, are reported.

One case of the Western type, an accidental laboratory infection, is described in detail.

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REPORT ON MEDICAL PROGRESS

UROLOGICAL SURGERY: THE URETHRAL CATHETER*

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DURING the last few years, especially since extensive employment of the transurethral resection of the prostate came into vogue, the whole problem of catheter drainage of the bladder has been receiving increased attention. This is due to the initiation or increase of infection of the urethra and bladder, which has been considered an inevitable complicating factor following the use of the inlying catheter. Such infection, even though mild, is always undesirable, but in cases in which the prostate has been removed either by the suprapubic or, better, by the perineal route, the outlet of the bladder is likely to be more thoroughly freed of all obstructing tissue than when the transurethral approach is used. Following complete freeing of the bladder outlet, the cystitis that remains after healing has taken place is usually of moderate severity only, and responds quickly to lavage of the bladder with antiseptics. Or it may occasionally be overcome spontaneously after a few weeks. On the other hand, infection of the bladder following a transurethral prostatectomy not infrequently increases in severity, so that in some cases the patient is found to have exchanged his symptoms of obstruction for the more disagreeable and uncomfortable ones of frequent and painful urination. And if there still remains a residual urine, only with great difficulty can the bladder be made sterile again by postoperative lavage or by drugs.

That this is not a necessary sequel of transurethral prostatectomy is evidenced by the experience of Thompson and Buchtel,[1] who find that after sufficient hyperplastic prostatic tissue has been thus removed, patients tolerate the postoperative inlying catheter well without evidence of increasing urethritis and cystitis. Others have been less fortunate, however, so that one often finds the expected benefits vitiated by persisting infection.

Because of infection, therefore, we find a general tendency today to subject patients to catheter drainage of the bladder before operation less frequently and for a much shorter time than formerly. In point of fact, in 96 out of 200 cases

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analyzed by Thompson and Buchtel no preliminary drainage was instituted, even though the blood urea nitrogen at the time of operation was 50 mg. per 100 cc. in 16 per cent of the patients. That so large a proportion of patients could undergo successful removal of the prostate by whatever method without preliminary drainage of the bladder reflects the fact that patients today are being referred for operation at an earlier stage of their difficulties, when the renal function is still intact or only slightly impaired. This is as it should be. The experience of these authorities also points to the fact that drainage of the bladder after operation, if there be no complicating infection, is often sufficient to relieve the embarrassment of the kidneys quite as efficiently at this time as when catheter drainage is employed as a preoperative step.

But in spite of newer knowledge and experience as to when the use of the catheter may be avoided, it is nonetheless an invaluable instrument demanding frequent use. Further studies have been made by Schulte[2] as concerns the bacteria to be found in the normal urethra of man. Of the four groups of patients chosen for study, none had had any previous infection of the urinary tract and none had any symptoms referable to it at the time of examination. In the first group, after cleansing of the glans with alcohol the second portion of the voided urine was examined bacteriologically. The organisms found in order of frequency were: micrococcus, diphtheroids, *Streptococcus faecalis,* alpha streptococcus, gamma streptococcus, *Staphylococcus albus, Escherichia coli, Aerobacter aerogenes* and pseudomonas. From a second group the urine was collected by catheter, with the same result except that the number of colonies was somewhat smaller. In the third group the second portion of the voided urine was cultured after the urethra had been gently irrigated with a solution of potassium permanganate. Half these cases still showed the micrococcus. In this group the prostatic secretion was studied as well, and was found in practically all cases to contain an alpha streptococcus. Schulte concludes, therefore, that the micrococcus is a normal inhabitant of the anterior urethra, while the alpha streptococcus is normal for the posterior urethra and prostate. A fourth group of patients, consisting of an approximately equal number of men and women, were studied at cystoscopy. Here also organisms were present, so that Schulte concludes that the urine from the bladder contained urethral bacteria, and that it is impossible to pass a cystoscope into the bladder through the urethra without contaminating the urine. While most clinical urologists will disagree with this last statement, basing their opinion on the many sterile cultures of the urine obtained in the routine examinations of the clinic, nevertheless the extreme prevalence of bacterial organisms in the male urethra is again emphasized by this study.

Granting the above, and in view of the fact that the use of the catheter is frequently imperative, the problem becomes one of ways and means to prevent increase of infection during such time as its employment is necessary. Basing his conclusions on the undoubted fact that trauma to the urethral walls caused by the catheter plays a large role in promoting growth of bacteria already present, Middleton[3] presents an argument favoring abolition of the retention (inlying) catheter in the preparation of all patients for prostatic surgery. He compares the transurethral enucleation of the prostate through a highly inflamed, edematous urethra to removing a cataract in the presence of acute conjunctivitis. Therefore, to avoid this urethritis he advocates intermittent or interval catheterization in the preoperative preparation of all patients. A male nurse or specially trained orderly is instructed to empty the patient's bladder by catheter at three- or four-hour intervals. A soft-rubber catheter (No. 16 Fr.) bearing a coudé olive-tip is inserted by the use of a sterile clamp and is not touched by the operator's hand. The objection that relief of embarrassed renal function may not be so prompt and definite by this interval catheterization as when the bladder is kept continuously empty by the inlying instrument, Middleton answers by noting that in normal life the bladder is emptied only intermittently. The rare case in which the catheter cannot be passed without a stilet, or in which bleeding follows its use, he still treats by continuous drainage.

In reply to Middleton's advocacy of the intermittent type of catheterization, Vose[4] stresses the value of the continuous catheter, when strict attention is paid to surgical cleanliness, to the use of a small, non-irritating size of catheter and to the elimination of occlusive strapping at the meatus. He maintains sterility by an entirely closed system of tubing, and a closed irrigation system when this is needed. He believes that the small catheter (No. 12 to No. 16 Fr.) allows opportunity around itself for drainage of any urethral products of inflammation at the meatus by not overdistending the urethra. Cumbersome adhesive-plaster holding devices have been eliminated by the use of a self-retaining catheter of the Foley type.

Recognizing the various drawbacks to both intermittent and continuous catheterization, and basing our argument on the fact that for the best and most consistent surgical end results, elimination of infection of whatever degree is imperative, we adopted at the clinic of the Peter Bent Brig-

ham Hospital about two years still another method of using the catheter for preoperative treatment of patients with prostatic retention. This method consists in introducing the catheter into the bladder just above the symphysis by the aid of a trocar and cannula. Thus the urethra is spared, while the more extensive suprapubic wound of the former two-stage prostatectomy is avoided. That the method endangers the peritoneum is true only when employed by the grossly inexpert. In point of fact the procedure is one at which the average house officer becomes quickly adept, familiar as he perforce is today with the frequent use of varieties of needles and trocars for parenteral medication. In the report of our experience with this method of bladder drainage by Austen[5] there is cited no case of injury to the peritoneum, and only two in which feared leakage of urine into the prevesical space made subsequent open suprapubic cystotomy advisable. The details of the introduction of the catheter are given by Austen as follows: The bladder must be tensely filled so as to be easily palpable above the symphysis pubis. If this is not already the case a urethral catheter should be introduced and the bladder filled to capacity. After shaving and sterilization of the operative field an area of local anesthesia is produced in the midline. In this area a small vertical incision 0.5 to 1 cm. in length is made through the skin and fascia just above the symphysis. The long needle of the apparatus devised by Kreutzmann[6] is plunged inward and downward behind the symphysis. When the bladder is entered, urine immediately trickles out through the needle. Should this not occur, the needle must be reinserted. The trocar and cannula are then passed over the needle, and by a slight rotary pressure the entire instrument enters the bladder guided by the needle. The needle and trocar are next removed and a No. 18 Fr. graduated catheter of soft rubber is introduced through the cannula. The cannula is then withdrawn, leaving the catheter in place for drainage. A single silk suture through catheter and skin fixes it at a level of about 12 cm. from the surface, and the outer end of the catheter is connected to a sterile drainage system.

In an editorial Creevy[7] discusses the care of the postoperative retention of urine. Three factors are held responsible for this retention: the dorsal position of the patient, the painful musculature of the abdominal wall and drugs. Creevy finds a definite tendency of opiates to increase the tonus of the internal vesical sphincter, and also to make the patient unaware of the fullness of his bladder until it is overdistended. Some surgeons withhold catheterization until prolonged painful distention of the bladder has rendered the situation intolerable. Such distention causes mucosal and submucosal hemorrhages, together with loss of tone to the detrusor muscle and subsequent vesical infection.[8] Note should always be made of the patient's fluid intake. It is entirely illogical to order a liter of fluid to be given intravenously just after operation, and at the same time to insist that catheterization, if necessary, be postponed at least eight hours. If retention persists so that more than three or four catheterizations are needed, Creevy advises that the instrument be made indwelling and connected to one arm of a sterile glass Y tube by rubber tubing, attached to a sterile reservoir hung at the bedside and containing sterile or antiseptic fluid. The other arm of the Y tube goes to a covered sterile empty bottle beneath the bed. This avoids the contamination inevitable when the bladder is irrigated by hand. The indwelling catheter should not be removed until the patient has gained strength enough to move about in bed. After its removal, the bladder should be tested several times for residual urine. In cases showing loss of bladder function due to lesions of the nervous system, Creevy first empties the bladder by the Credé method at more or less frequent intervals. If this is not possible, he institutes tidal drainage after the method of Munro.[9]

A series of 84 general surgical cases in which urinary retention occurred after operation has been studied bacteriologically by Stalker and Schulte.[10] None of the patients had had previous urinary infection. In the first group of 73 patients the catheter was used intermittently to relieve the retention. The second group consisted of 11 patients put on constant urethral drainage at the time of operative resection of the rectum for cancer. As a result of this study the authors find that patients with postoperative urinary retention following general surgical procedures are best treated by frequent intermittent catheterization, if possible. This can usually be carried out for a period of two to four or more days without the development of evidence of urinary-tract infection. When the inlying catheter has been used, infection invariably develops in twenty-four to thirty-six hours. Once bacteria have developed, they increase rapidly in number while the catheter is retained. When intermittent catheterization is employed, the increase is not so rapid, and in some cases even a decrease is noted. In many cases in which only a few bacteria are present in the urine, the patients have no clinical symptoms of infection of the urinary tract. Frequently these bacteria gradually disappear without treatment, and for this reason the presence of a

positive culture alone is not sufficient to warrant instituting therapy. However, when the number of bacteria increases rapidly, and especially when this increase occurs where residual urine is present, clinical symptoms of infection are almost certain to occur, and therapy is required. In the group of patients maintained on indwelling catheters, prophylactic treatment was instituted by the use of sulfanilamide, 1 gm. daily, from the time of operation until voluntary urination was resumed. The resulting concentration of sulfanilamide in the urine — 35 mg. per 100 cc. — was sufficient to render the urine bacteriostatic. The writers believe that in their experience such prophylactic use of sulfanilamide has been definitely beneficial, for while it does not suffice to keep the urine free of bacteria in every case, it keeps down the number of bacteria and prevents the development of clinical symptoms.

Our present attitude toward the catheter and its use can therefore be summed up somewhat as follows:

It is an indispensable instrument, but its use should always be attended by extreme aseptic precautions.

The urethra, in both men and women, normally harbors bacteria of various sorts which cannot be eradicated permanently.

The inevitable trauma, though slight, following the passage of a catheter through the urethra, or especially that following its permanent use, permits increased growth of these bacteria, with resultant urinary infection — always of the bladder, sometimes of one or the other kidney.

The use of the indwelling catheter should be accompanied by the creation of a bacteriostatic urine through the administration of sulfanilamide, as well as by daily or twice daily irrigation of the bladder with an actively antiseptic solution.

An increasing recognition of the dangers attending the use of the catheter in the urethra is evident. This recognition has taken the form on the one hand of a definite increase in the number of patients who are subjected to prostatectomy without preliminary drainage of the bladder, and on the other hand, when the catheter must be used, of the introduction of it by the method of suprapubic puncture, thus avoiding the urethra.

Renewed emphasis is found of the importance of careful postoperative attention to the bladder in order to prevent its overdistention.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 26401

PRESENTATION OF CASE

A forty-two-year-old man was admitted to the hospital complaining of attacks of postprandial, precordial pain, and dyspnea on exertion.

Eight weeks before admission the patient developed what he called "indigestion pains" following meals, located high under the left lower ribs. These non-radiating pains occurred in attacks, lasting a few minutes, and were usually the result of hurried eating and also produced by physical exertion. Following one of these attacks, he developed dyspnea, which forced him to sit down. During the eight-week interval before admission, he fainted two or three times; each time there were no prodromal symptoms, and he remained unconscious for from two to five minutes. Six weeks before entry orthopnea appeared, which on a few occasions forced him to sit upright in a chair throughout the night. He stopped work and remained in bed most of the time. Three and again two weeks before admission he developed ankle edema, and a fear of the ingestion of food because of the vomiting it engendered. One week before entry he was seen by a physician, who prescribed a "pill" of digitalis three times a day; he had taken a total of 15 pills at the time of entry.

About one year before entry the patient badly injured the left fourth and fifth fingers. An attempt was made to save one finger; two surgical operations were performed on it, but they were unsuccessful. It was stated that during this time the patient suffered pains that he often described as radiating up the arm to his "heart." He had had pneumonia twice nineteen years before admission. Tonsillectomy and adenoidectomy had been performed fourteen years before admission. He gave no history of rheumatic fever or scarlet fever. The family history was negative.

Physical examination revealed an orthopneic, acutely ill, cold and clammy man whose neck veins were markedly distended and who displayed Cheyne-Stokes respirations. The pupils were equal but reacted sluggishly to light. The tongue was red and slightly smooth. The throat was injected. The heart was enlarged, the sounds were distant and of poor quality, and there was a pronounced gallop rhythm. There were no murmurs, and the blood pressure was 135 systolic, 100 diastolic. Rales were prominent throughout the chest; the breath sounds were dull and distant at the bases, especially on the right. The abdomen was distended with fluid, and the liver was felt 4 to 5 cm. below the costal margin. The tip of the spleen was questionably palpable. The lower legs were edematous. The left fourth and fifth terminal phalanges were missing.

The temperature was 97.6°F., the pulse 100, and the respirations 25.

Examination of the urine was negative. A blood Hinton test was negative. The blood nonprotein nitrogen was 25 mg. per 100 cc. An electrocardiogram showed a PR interval of 0.21 second, with partial auriculoventricular block and a rate of 90. T_1 was low, T_2 and T_3 slightly inverted. The QRS complexes in Leads 1, 2 and 3 showed low voltage; those in Leads 2 and 3 were slurred and notched. R_4 was small. An x-ray film of the chest, taken with a portable machine, revealed a hazy, mottled dullness scattered over both lung fields. There was an old fracture of the left ninth rib, with an overgrowth of callus. The heart was prominent in the region of the left ventricle; the aorta was tortuous.

On the day after admission, the patient's temperature, pulse, and respirations rose to 99.8°F., 120 and 36, respectively. He became restless and delirious. He was quieted with sedatives, and the daily administration of digitalis was continued. The blood pressure on the fourth day was 105 systolic, 60 diastolic. He was given oxygen therapy and mercurial diuretics. On the fifth day, however, he began to fail, and that evening he very suddenly developed labored respirations and quickly expired.

DIFFERENTIAL DIAGNOSIS

DR. PAUL D. WHITE: This man was relatively young to have had so much heart disease of the coronary type. At forty-two years of age, he may, however, have belonged in the rheumatic group.

The description of the precordial pain is not detailed enough. We may get more information later. We should like to know which came first on exertion, the precordial pain or the dyspnea. The pain could have been due to indigestion alone and as described is not diagnostic. The dyspnea on exertion cannot, however, be ascribed to indigestion; it might have been due to pulmonary disease or to myocardial weakness. If we had questioned him closely we might have found that the so-called "dyspnea" on exertion was really substernal oppression, that is, angina pectoris and not actual dyspnea. Such an error in interpretation is common. Hence I am a little doubtful about that symptom.

We learn that effort as well as eating induced

these pains that are not adequately described. It is characteristic of coronary insufficiency that angina pectoris is induced less by eating than by effort, but more by effort after eating than by effort on an empty stomach. If angina pectoris is induced by eating alone the coronary insufficiency is of high degree, and must be considered very serious. When very slight effort induces angina pectoris that fact too is an indication of severe coronary insufficiency. "Following one of these attacks, he developed dyspnea, which forced him to sit down." If dyspnea is associated with rapidly progressive coronary disease then there must also be cardiac dilatation and failure. We do not see congestive heart failure from coronary disease alone unless there is actual occlusion, usually with myocardial infarction. We do not encounter coronary disease with appreciable cardiac enlargement unless there is actual thrombosis and infarction, but in such cases the heart can get very large without hypertension.

There is no statement as to whether he had convulsions during syncope — probably not. Apparently he had not fainted before this. On top of the other symptoms this is strongly suggestive of Adams–Stokes attacks, which are due primarily to the effect of extensive coronary disease on the conduction system of the heart. Apparently these were not just nervous fainting spells. We do not really know, however, whether the syncope was due to collapse from fatigue or hunger or whether he was having a real Adams–Stokes syndrome or even an excessive reaction to angina pectoris, the so-called "syncope anginosa."

"Six weeks before entry orthopnea appeared which on a few occasions forced him to sit upright in a chair throughout the night." That makes the dyspnea significant, and means that we can rule out asthma and other such factors, and that there was myocardial failure, that is, left ventricular failure, which can quickly follow coronary occlusion even though there has been no prolonged pain, as in this case.

". . . he developed ankle edema. . . ." The right ventricle was doubtless failing, although he might have had a bilateral leg phlebitis to explain such ankle edema. If the edema were one sided we might say phlebitis, but on the basis of bilateral edema and the previous history of dyspnea we should diagnose failure first of the left ventricle and then of the right. "He was seen by a physician one week before entry, who prescribed a 'pill' of digitalis three times a day. . . ." This was seven weeks after his illness began. The digitalization was very mild.

A big question is, Why did he have these pains radiating up the arm to the heart? Were they associated with the finger injury or did they come as the result of walking uphill. In other words, he might have had some angina pectoris for a year before this final illness. Is there any statement about what activity produced the pain? The history is very vague on this point. Angina pectoris could start in this way as well as in any other way. The paragraph about the past history seems wholly unimportant unless these pains in the arm were brought on by effort.

In the physical examination we find gallop rhythm, heart sounds of poor quality, and slight diastolic hypertension which perhaps is not important and might have been temporary. Rales were prominent not just at the bases but generally — perhaps due to pulmonary edema or emphysema. The dulling of the breath sounds was undoubtedly due to hydrothorax.

We have not enough evidence to indicate a bacterial endocarditis complicating the picture. The spleen may have been congested, although we do not usually feel it in case of simple congestive heart failure.

"The temperature was 97.6°F." If he was dyspneic, we should know the rectal temperature. He may have had pulmonary infection or infarction.

The electrocardiogram backs up the suggestion of the Adams–Stokes attacks previously mentioned, although not strongly. It is also in accord with the diagnosis of posterior myocardial infarction, since heart block and changes in T_2 and T_3 are quite characteristic of early infarction due to occlusion of the right coronary artery or the circumflex branch of the left coronary artery supplying the basal portion of the heart. Low voltage is a characteristic coronary-disease finding. The QRS complexes are typically coronary. With low voltage in a normal person you do not have changes in the T waves. Also there was a very small R wave in Lead 4. Heart block of severe grade in coronary disease due to thrombosis of the right coronary artery or the circumflex branch of the left coronary artery may be temporary. I have seen it last a day or two, and then disappear, with a return to normal rhythm.

There was not much fluid at the lung bases, according to the x-ray film. There was increase in the hilar markings of the lungs. The heart was large, deep as well as wide. There was no marked bulging of the pulmonary artery or marked prominence of either auricle.

Although the diastolic blood pressure went down from 100 to 60, he certainly was not any better. Whether this drop was associated with failure I do not know. He may have had further difficulties in the circulatory state as the result of pulmonary complications.

Terminal pulmonary infarction or infection is the best bet as a part of the final story during the last day or two. The whole illness of about two and a half months raises a strong suspicion of coronary involvement, with rapid progression. The cardiac death suggests a sudden accident and not hypertensive or rheumatic heart failure, cardiovascular syphilis or some very rare kind of heart disease, although they are possibilities. There is no evidence of a congenital abnormality. The case suggests coronary disease, even though the onset is not the usual one for coronary thrombosis, nor an absolutely typical one for angina pectoris. The history of symptoms is not, however, adequate. The electrocardiogram suggests basal myocardial infarction and failure of the myocardium, and the early death suggests infarction or infection of the lungs. I cannot see that there is anything but coronary disease from beginning to end, except for a terminal complication, and I should expect to find an infarct at the left lung base.

Dr. Howard B. Sprague: I saw the patient with Dr. William B. Breed. It was difficult to get a history because he was so sick, but I made the note that he probably had been dyspneic for two years because he told me he had had trouble in going up stairs during that time. I was unable to fit the case into any very clear category. My note was as follows:

> Very interesting and, to me, obscure case. Rheumatic heart disease seems out, as does syphilitic heart disease, unless it is an acute myocarditis. He has probably been dyspneic for two years. The heart is enlarged, and there is a gallop rhythm. The electrocardiogram shows no evidence of previous hypertension. Coronary occlusion is not proved, and there is no evidence for pericardial disease. If this is not a case of diffuse coronary disease, as suggested by the low-voltage electrocardiogram, it is probably the rare one of a big heart of unknown etiology which cannot be diagnosed even at autopsy.

Dr. White: I may be making too much of the fainting attack and the solitary prolonged PR interval. I do not recall whether fainting occurs in heart disease of unknown etiology. I may be paying too much attention to the symptoms of heart pain and syncope.

Clinical Diagnoses

Cardiac hypertrophy, idiopathic.
Congestive failure.

Dr. White's Diagnoses

Coronary heart disease, with posterior (basal) myocardial infarction.
Cardiac enlargement.
Congestive heart failure.
Pulmonary congestion and infarction (terminal).

Anatomical Diagnoses

Cardiac hypertrophy and dilatation (idiopathic?).
Cerebral infarct, right occipital lobe.
Pulmonary embolism.
Pulmonary infarction, right.
Cortical infarct of kidney, recent.
Thrombosis of popliteal vein.
Chronic passive congestion: lungs, liver, spleen and kidneys.
Hydrothorax, bilateral.

Pathological Discussion

Dr. Benjamin Castleman: The heart weighed 425 gm. It was not so hypertrophied as one might expect, but the right auricle and ventricle were dilated to about twice normal size. Examination of the myocardium showed no evidence of infarction or even fibrosis. The cavities contained no mural thrombi, and the valves were perfectly normal. We examined the coronaries very carefully but found no significant amount of coronary disease. There was a little atheroma here and there, but certainly not more than one would expect in a man of forty-two. Microscopic examination of the myocardium did not add very much. There was very little scarring. The prominent feature of the microscopic examination was the marked fibrosis and thickening — in places up to a millimeter in depth — of the endocardium. This endocardial lesion has been described occasionally in congenital idiopathic hypertrophy in infants. We looked very carefully for Aschoff nodules and other stigmas of rheumatic disease in the myocardium and could not find any. So far as the heart is concerned we certainly can say that it does not represent a case of coronary or rheumatic heart disease.

Dr. White: Did the microscopic examination of the myocardium show fibrosis?

Dr. Castleman: Practically none. We cut several sections of the heart muscle in various places, and the only positive finding was the scarring of the endocardium.

Dr. White: On both sides?

Dr. Castleman: No; almost all on the left side. We also hunted for glycogen in the myocardium and found only a normal amount, certainly not what you would see in von Gierke's disease. The lungs showed chronic passive congestion and one small, fairly old infarct in the right middle lobe. The source of the embolus that caused this infarct was a thrombus in the popliteal vein. There was also an old infarct in the left kidney, which if I recall correctly was also true of some of these cases of cardiac hypertrophy

of unknown etiology in adults that Levy* reported. I believe he found small thrombi in the interstices of the heart muscle as the source of the emboli.

DR. WHITE: What was the thickness of the heart wall?

DR. CASTLEMAN: The right was 5 mm., the left 15 mm.; this points to a little hypertrophy on both sides. I do not believe that we can rule out hypertension, since he was in heart failure and we do not know what the blood pressure was before admission.

DR. WHITE: It was not a big enough heart for hypertension alone.

DR. CASTLEMAN: At least one of the fainting spells can be explained by the presence of a recent large infarct in the right occipital lobe of the brain. In addition, recent small hemorrhages were scattered over the surface of the brain.

DR. SPRAGUE: The pulmonary circulation was entirely negative as I remember. You ruled out intrinsic disease there?

DR. CASTLEMAN: Yes.

CASE 26402

PRESENTATION OF CASE

A seventy-nine-year-old man entered the hospital complaining of shortness of breath.

The patient was first seen in the Out Patient Department where he stated that he had noticed an increasing shortness of breath, accompanied by substernal constriction on exertion, for the past six months. In the past three months he had been compelled to rest frequently while walking. During the last few weeks he awakened at night dyspneic and unable to take a full breath; this was relieved by sitting up. In addition the patient had had an aching pain in the region of the left sacroiliac joint which radiated down the left thigh and was unrelated to motion. This had been present for two years and had increased of late to the extent that it required "dope" for relief. In general he felt weak and had lost 19 pounds in the last four months.

In the past he had suffered from gonorrhea at eighteen and typhoid fever at fifty-four years of age. Urethral strictures had been dilated by sounds two years before. His father died at sixty-eight of "kidney trouble," and his mother of tuberculosis at forty.

On physical examination the patient was alert and appeared well. The heart was enlarged to the left anterior axillary line. The rhythm was regular, the sounds of good quality; no murmurs were heard. The blood pressure was 180 systolic, 95 diastolic. There were a right inguinal hernia and diastasis recti abdominis. A firm, tender, rounded and fusiform mass about 6 cm. in diameter was palpable in the left mid-abdomen. It pulsated and did not move with respiration. Rectal examination was negative, and the prostate not enlarged.

The temperature and respirations were normal, and the pulse between 80 and 100.

Examination of the urine was negative. A white-cell count was 10,000. A blood Hinton test was negative.

X-ray films of the chest showed the heart to be enlarged in the region of the left ventricle. The abdominal aorta was extensively calcified, and the calcium appeared to expand in a fusiform bridge between the first and fourth lumbar vertebrae to the left of the spine. Electrocardiographic recordings showed partial auriculoventricular block, blocked premature auricular beats and nodal escape. Lumbar puncture and cerebrospinal-fluid Wassermann tests were negative.

The patient was placed on digitalis and aminophyllin. He improved under this treatment and when he restricted his activity remained free from dyspnea and substernal distress. The seventh week after he had come under observation he began to have attacks of left-upper-quadrant pain of unstated character, associated with shortness of breath and requiring a hypodermic for relief. The only addition to the clinical findings at this time was pitting edema of the ankles. One month later he was admitted to the hospital for observation and study of the abdominal mass. The day after entry the patient walked into the lavatory and was found dead a few minutes later.

DIFFERENTIAL DIAGNOSIS

DR. HOWARD B. SPRAGUE: This man survived almost to the age of eighty years, so that he probably succumbed to the changes that are due to the passage of time, and one thinks at once of arteriosclerosis and cancer. He had a relatively short history of what appears to be cardiovascular difficulty with shortness of breath. It is a little difficult to be sure whether this shortness of breath can be called evidence of congestive failure or whether the breath was cut off when he exerted himself and therefore was an anginal manifestation. However, there was substernal constriction on exertion, a fact that suggests angina pectoris. He also was having nocturnal attacks of dyspnea, relieved by sitting up, and finally pain in the left sacroiliac region radiating down the left leg. The story of weight loss at this age is not necessarily of any significance except that it may go with arteriosclerosis. He had had gonorrhea when he was young; he had had typhoid fe-

*Levy, R. L., and Rousselot, L. M.: Cardiac hypertrophy of unknown etiology in young adults. *Am. Heart J.* 9:178-196, 1933.

ver. Typhoid fever has been blamed by some physicians for subsequent vascular changes, but if it was a factor, it took a long time with this man. The examination showed that he was alert and appeared well, an observation that is quite consistent with the story that his troubles were largely of a paroxysmal or episodic nature. He had to exert himself in order for symptoms to appear, or be awakened in the night with symptoms. He had a large heart extending to the left axillary line. The rhythm was regular, and the sounds of good quality; there were no murmurs. There seems to have been no evidence of valvular disease. He had moderate systolic hypertension, and a mass in the abdomen.

You have heard the report of the x-ray examination. You can see the calcification that was described in the wall of the aorta. The heart does not appear so large as the physical examination suggested. There is nothing suspicious in the thoracic aorta.

Dr. Paul D. White: Is the aortic calcification in the region of the palpable tumor?

Dr. Sprague: I judge that it was because it extends from the first to the fourth lumbar vertebra and it was noted that the tumor was in the mid-abdomen. It was just about opposite this point, which I should judge to be the position of the umbilicus, and the dilatation would extend from the level of the ribs to that of the ilium.

The electrocardiogram is abnormal chiefly because of the occurrence of partial auriculoventricular block. All attempts to demonstrate serological evidence of syphilis in this man were unavailing, including a blood Hinton test and a spinal-fluid Wassermann test. He improved under the treatment but entered the hospital to have the tumor mass studied. He showed at that time some pitting edema of the ankles, which might be further evidence of congestive failure, although it might also have been due to some obstruction higher up in the veins or lymphatics interfering with drainage from the legs. He died very suddenly.

The question that concerns us chiefly, I think, is what the abdominal mass had to do with his death. You remember last week we had a case of syphilitic aortic aneurysm showing marked calcification in its walls. They certainly describe here what appears to be a dilatation of the abdominal aorta with calcification in the walls. It is a fusiform affair, not saccular. It seems to me, however, it could be responsible for the referred leg symptoms and conceivably the cause of the vague attacks of left-upper-quadrant pain. The calcified fusiform bridge at the upper end of the mass was apparently in or close to the left upper quadrant, and this mass extends nearly the whole length of the side of the abdomen and could cause pressure, perhaps involving several nerve pathways. There does not seem to be any evidence by x-ray of erosion of any of the vertebrae.

I think that we shall have to consider that this is an aneurysmal dilatation of the aorta, and the question is whether it is on a syphilitic or on an arteriosclerotic basis. Abdominal aneurysms are difficult to diagnose and have been picked up here at autopsy entirely unsuspected and causing no symptoms. So far as the syphilitic background is concerned I might give you some figures that I collected this spring about our experience here with aneurysm of the aorta. In 28 cases of aneurysm of the aorta proved to be syphilitic at autopsy between 1908 and 1938, there were 15 in which Wassermann tests alone were done and in 3 of these the test was negative; there were 5 in which the Hinton test alone was done, and in 2 of these it was negative; and there were 8 in which both tests were done, and none of these would have been missed. In a series of 23 private cases of syphilitic aortitis 20 had positive serological tests. We have here two types of test,—blood and spinal fluid,—and both were negative. We have an old man who gave us a story of gonococcal infection, and yet we can only guess about any syphilitic background, with possible calcific changes. He had, I believe, hypertensive and coronary heart disease with angina pectoris. He died suddenly, and we need no other diagnosis to explain the sudden exitus.

We have not, it seems to me, sufficient evidence to suspect that there was a rupture of the dilated abdominal aorta, although the history just before the patient was admitted to the hospital included pains in the left upper quadrant that suggest some stretching or early subacute rupture of a dilated aorta. I am going to guess, then, that he showed coronary arteriosclerosis and that he probably died of angina pectoris, in which case one would not necessarily expect to find a fresh thrombus occluding a vessel. He may very well have had fluid blood in the coronary arteries. There was a diffuse arteriosclerotic process in the aorta. I should not be surprised if he had more than one aneurysmal dilatation in the aortic tree or even in the large vessels. I vote against syphilis in this case, and I find no reason to suspect that this was a dissecting aneurysm such as we see in the thoracic aorta, for the reason that he gave no story of the acute attack of pain that would have accompanied a true dissection and because dissecting aneurysm is a disease of the thoracic aorta which dissects into the abdominal aorta.

DR. BENJAMIN CASTLEMAN: Dr. White, have you any comments?

DR. WHITE: No, except to say that the death in the lavatory is a very important point so far as the hazard of constipation is concerned in a patient with angina pectoris. I wonder if there was any story of straining at stools. Was there any note of constipation?

DR. SEDGWICK MEAD: Not that I recall.

DR. WHITE: I have occasionally advised such patients if they are constipated to take tablets of nitroglycerin just before defecation.

DR. CASTLEMAN: The commonest cause of death occurring in the lavatory in this hospital is massive pulmonary embolism.

DR. MEAD: The complaint that this patient had on entry to the hospital was a very severe pain radiating down the lateral aspect of the left leg. The cardiac symptoms were more or less under control from the previous digitalization. The abdominal mass was distinctly more prominent on the left than on the right; it was high enough so that a surgical consultant thought that it was perhaps an aneurysm of the splenic artery.

CLINICAL DIAGNOSES

Ruptured, arteriosclerotic, abdominal aortic aneurysm.
Hypertension.
Hypertensive arteriosclerotic heart disease, with coronary sclerosis.
Angina pectoris.
Right inguinal hernia.

DR. SPRAGUE'S DIAGNOSES

Hypertensive and coronary heart disease.
Coronary sclerosis, with angina pectoris as cause of death.
Arteriosclerotic abdominal aneurysm, with questionable subacute perforation.

ANATOMICAL DIAGNOSES

Aneurysm of abdominal aorta, syphilitic, with rupture.
Retroperitoneal hematoma and ecchymoses.
Hemoperitoneum.
Arteriosclerosis, marked aortic and coronary.
Coronary occlusion, old.
Cardiac infarct, localized, healed?
Cardiac hypertrophy, hypertensive type.
Nephritis, chronic vascular.
Emaciation, slight.
Prostatic hyperplasia.
Diverticulosis coli.
Perisplenitis, chronic.
Cholecystitis, chronic.
Cholelithiasis.

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: At autopsy we found a heart that weighed 500 gm. and showed no disease of any of the valves. The coronary arteries were sclerotic and calcified, the lumen of the main right branch being reduced to a pinpoint opening by an old thrombus. There was patchy scarring of the myocardium, with one fairly large scar that apparently was the result of the old thrombotic process.

DR. WHITE: Where was that?

DR. CASTLEMAN: That was on the posterior wall of the left ventricle near the septum. If the autopsy were limited to just those findings we would have concluded that the man died of angina pectoris. However, abdominal examination showed rupture of a tremendous aneurysm which rose from the anterior wall of the aorta 7 cm. above the bifurcation. The aneurysm itself measured about 8 cm. in length and contained an organized mural thrombus. At the time of autopsy we were all agreed it was probably an arteriosclerotic aneurysm because of the marked calcification. Sections, however, showed fairly good evidence of syphilis — lymphocytic infiltration around the vasa vasorum of the media and adventitia, and destruction of elastic tissue. The ascending aorta was arteriosclerotic throughout.

The blood that had escaped from the aneurysm formed a hematoma in the retroperitoneal tissues, which in turn had ruptured into the peritoneal cavity and pelvis.

DR. WHITE: I should like to add another word with respect to my recent experience with arteriosclerotic thoracic aortic aneurysms in aged people. Abdominal arteriosclerotic aortic aneurysms have been much commoner than have thoracic aneurysms. My attention was called to the thoracic type a few years ago because of an experience with two very old ladies both of whom had been known to have aneurysmal dilatation of the aorta with no evidence of syphilis and both of whom died of rupture of the aneurysm, one with dissection and one without dissection but with multiple aneurysms. Autopsy confirmed the arteriosclerotic nature of the aneurysms in both cases.

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UNITED STATES MARINE HOSPITAL AT THE PORT OF BOSTON

A new chapter in a long and honorable history was written on Thursday, June 7, 1940, when the sixth home of the United States Marine Hospital, the oldest hospital in Massachusetts, established in 1799, was dedicated in Boston. For almost one hundred and forty-two years this establishment has furnished medical care to seamen and, in recent years, to government employees not members of the uniformed services. Dr. Thomas Parran, surgeon-general of the United States Public Health Service, and the Honorable Paul V. McNutt, federal security administrator, addressed the gathering at the dedication, both pointing out in their remarks that the public-health program of the federal government was in its own way as important as the national defense program.

To many the Marine Hospital has been, as a matter of fact, frequently confused with the Naval Hospital whose neighbor it was in Chelsea. An act of Congress, signed in July, 1799, by Massachusetts's own John Adams, provided for the "relief of sick and disabled seamen," and the first marine hospital in the United States was opened in temporary quarters on Castle Island (then Castle William) in South Boston. Only three hospitals in the United States are older than the Marine Hospital at the Port of Boston — the Pennsylvania Hospital at Philadelphia (1752), the Charity Hospital of Louisiana at New Orleans (1784) and the New York Hospital at New York City (1791).

The masters of sailing vessels were, in the early days of the nineteenth century, authorized to deduct certain sums each month from the pay of seamen, which were paid into a fund used to support this and other marine hospitals along the Atlantic coast. Thus present-day plans for compulsory health and sickness insurance were anticipated by more than a century. The administration of these contributions and the general oversight of the Marine Hospital resided in the office of Secretary of the Treasury, where it remained until July 1, 1939 when it was transferred to the Federal Security Agency. The contributions by seamen were continued until 1884, when expenses so far outran contributions that the assessments were discontinued and the cost of hospital maintenance was met by levying a tonnage tax on foreign vessels. In a short time this tax was found to be greater than necessary, and the money was paid directly to the United States Treasury, which in turn paid hospital costs from its general funds.

All these facts and many others about the hospital's one-hundred-and-forty-two-year story can be learned from a pamphlet entitled *The United States Marine Hospital at the Port of Boston, 1799–1940,* by William John Trask, its medical director from 1935 to 1940. Benjamin Waterhouse, Hersey Professor of the Theory and Practice of Physic at Harvard Medical School from 1783 to 1812, was an early director, and under his leadership an outpatient department was initiated. Today this department is one of the most active in the hospital and provides treatment for almost seven times the number of patients who are hospitalized. The house patients in 1810 numbered 242, and in 1812 totaled 649, due to the casualties of the war

then being waged. British prisoners and seamen from the frigates *Constitution* (after her battle with the *Guerriere*) and *Congress,* from merchant vessels and from the clippers in the China trade, all received the finest care available at the time. In the early days it was the custom for sailors to be brought directly to the hospital, many in extremis because of the delay in treating injuries before hospitalization. Today the Coast Guard and Public Health Service, with the co-operation of the staff of the Marine Hospital, send advice in regard to the treatment of seamen aboard ship via radio telephone, and when the need is dire the patient is brought to the hospital by airplane or speedy motorboat.

For the accomplishments of his predecessors and their staffs Dr. Trask has spared no praise. For his own work he is overmodest. It was his keen insight into the problems of the hospital and its physical limitations when he took up his duties, as well as his earnest planning, that is chiefly responsible for the new plant at Brighton. Dr. Grover Kempf, well known for his experience in public-health administration, has been selected to succeed Dr. Trask and is assuming the duties of directing this hospital in the new phase of its development. With an energetic and well-trained full-time staff and an excellent group of consultants who represent all the specialties, its services are not surpassed by the finest general hospitals in the country. In the early years of its existence the hospital was a teaching unit of Harvard Medical School, and this function is continued in a slightly different form at the present time, chiefly by means of intern training. In the past, research facilities have been necessarily limited by the demands for ward space; in the new building provision has been made for this purpose, and a number of specific research projects are being planned.

The United States Marine Hospital at the Port of Boston represents and typifies the "American way" in medicine: for nearly one hundred and forty-two years it has maintained a high tradition of service to men who go down to the sea in ships. With new facilities it looks forward to long continuance and expansion of its service as one of the leading, although not the largest, government-operated hospitals in the Western hemisphere.

CHILDHOOD MORTALITY

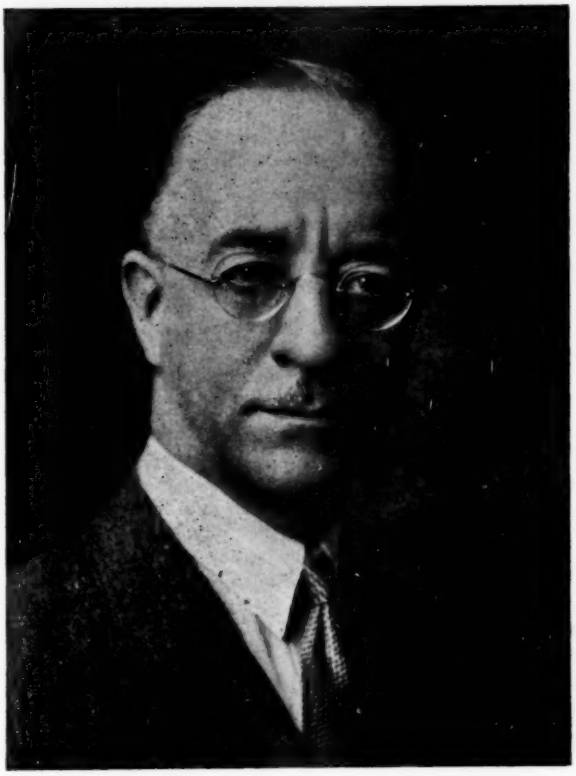
A RECENT *Statistical Bulletin* of the Metropolitan Life Insurance Company, comments favorably on the reduction of mortality in childhood during the last three decades. Today the mortality in the ages from one to fourteen years is only a fifth of that less than thirty years ago: in 1939 there were only 1.3 deaths per 1000 insured white children of these ages as compared with a rate of 6.4 in 1911.

A number of gains are responsible for this remarkable improvement. Influenza and pneumonia, heading the list in 1911, with a death rate of 100 per 100,000, rose to an all-time high during the pandemic of 1918 with a rate of 345 but dropped to less than 21 in 1939; the latter figure is below the rate for total accidents, and thus drops this cause of death from first into second place.

Diphtheria, second in the list in 1911 with a rate of 88 per 100,000 children, dropped to 4 in 1939. The other three principal communicable diseases of childhood registered similar, though less spectacular, drops: scarlet fever from 41 to 2, measles from 36 to 2, and whooping cough from 20 to 2. Diarrhea and enteritis were in third place in 1911, but the death rate of nearly 65 dropped to 4 in 1939; during the corresponding period the rate for tuberculosis fell steadily from 45 to less than 6.

Acute rheumatic fever, nephritis and organic heart disease dropped to a third or a quarter of their original rates; all fatal accidents fell from 57 in 1911 to 26 in 1939. Deaths from appendicitis have shown no improvement, remaining at about 10 per 100,000, and consequently this cause of death moved from eleventh place in 1911 to third place in 1939. Deaths from automobile accidents are the only ones to show an increase, — from less than 3 in 1911 to over 10 in 1939, — and they now stand second only to those from influenza and pneumonia. Even this figure, however, shows a substantial reduction from the maximum rate of 18 in 1929.

These figures are indicative of the slow but steady advances that humanitarian science has made in ameliorating the condition of mankind. It is a sad commentary on modern civilization that such hard-won gains can be so rapidly reversed by other causes.



ALEXANDER SWANSON BEGG
1881 – 1940

It is with a deep sense of loss that the *Journal* records the sudden death of the secretary of the Massachusetts Medical Society. For five years he had faithfully and effectively fulfilled the exacting and arduous tasks of this office.

Dr. Begg was best known, however, as an able contributor to the advancement of medical education. He enjoyed expending the major part of his unexcelled energy in this field. He started medical teaching at his alma mater, Drake University, and continued this activity at Harvard University and at Boston University, where he had been professor of anatomy for nineteen years. For the past seventeen years he was the energetic and respected dean of Boston University School of Medicine.

His activities outside of the medical school were many and varied. He was widely known for his two-year service in the Medical Corps of the United States Army during World War I. For eleven months he commanded Base Hospital 88 in France, having advanced from the rank of lieutenant to that of lieutenant colonel. Since then he participated actively in the Medical Reserve Corps in the New England area, holding the rank of colonel in the Auxiliary Corps, United States Army. In the past he served on the Executive Council of the Association of American Medical Colleges, as chairman of the Committee on Education of the Association of American Medical Colleges, as president of the Norfolk District Medical Society of the Massachusetts Medical Society and as a director of the Boston Chamber of Commerce and chairman of its Committee on Public Health. At the time of his death he was Massachusetts chairman of the Committee on Medical Preparedness of the American Medical Association, on the editorial board of the *Diplomate* and chief examiner of the Boston Subsidiary Board for the Part III examinations of the National Board of Medical Examiners.

He was a member of the American Association for the Advancement of Science, the American Association of Anatomists, the Association of Military Surgeons, the American Medical Association and Phi Beta Kappa.

Those who realize the significance of the distinctive ability with which the duties of these positions were fulfilled, as well as the many who were privileged to benefit intimately from his helpful guidance, generosity, good humor and friendliness, are convinced that Dr. Begg's leadership in medical education and other matters relating to the medical profession will not soon be forgotten.

MEDICAL EPONYM

Duroziez's Sign

Paul Louis Duroziez (1826–1897), of Paris, contributed an article, "Du double souffle intermittent crural, comme signe de l'insuffisance aortique [The Intermittent Crural Double Murmur as a Sign of Aortic Insufficiency]," to the *Archives générales de Médecine* (Series 5, 17:417–443 and 588–605, 1861). A portion of the translation follows:

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The maternal-mortality study[1–3] for the years 1937–1939 shows that in Massachusetts 64 deaths were attributed to pneumonia complicating pregnancy. This shows the seriousness of the condition. Pregnancy certainly increases the gravity of any case of pneumonia. From the obstetric standpoint it must be emphasized that pregnancy or labor should be looked upon as a mere incident and that the real hazard is the pneumonia. If pneumonia cases occurring during pregnancy are to be intelligently handled so that the maternal death rate is reduced, it must be remembered that pneumonia is a medical disease requiring hospitalization and consultation, that the obstetric handling of these cases is entirely conservative and that nothing should be done to stimulate the onset of labor.

The progress of the treatment of pneumonia based on serotherapy and chemotherapy has been brought out in these cases. Before serotherapy and chemotherapy were applicable to the treatment of pneumonia the death rate of patients with pneumonia during pregnancy was always much higher than that of those with pneumonia uncomplicated by pregnancy. Although a higher mortality rate in pregnancy cases will undoubtedly continue, it seems likely that the intelligent application of these two methods of treatment will result in a lowering of the rate that will correspond to that already obtained in uncomplicated cases of pneumonia.

As already stated, conservatism from the obstetric point of view is essential. Patients in the middle trimester of pregnancy frequently start in labor spontaneously; such labors should be allowed to proceed normally. If an anesthetic is necessary for delivery, spinal anesthesia is undoubtedly the method of choice. There have been those who have advocated the induction of labor in patients suffering from pneumonia: this procedure is to be condemned. If a patient at term develops pneumonia, if labor starts and if a cesarean section is clearly indicated, spinal anesthesia again is the safest and most satisfactory procedure that can be used.

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FRIDAY, OCTOBER 11

*9–10 a.m. Routine Office Cardiovascular Disease. Dr. S. A. Levine. Joseph H. Pratt Diagnostic Hospital.

cannot be given, it should be noted that the volume covers the anatomy and physiology of the hypothalamus as well as the clinical symptoms associated with dysfunction of this part of the brain. There is an excellent historical résumé of the subject by the chairman of the editorial board, Professor John F. Fulton, and Fröhlich's original paper on a tumor of the hypophysis, dated 1901, written in German and illustrated, is reprinted. It is interesting to know that Fröhlich's patient is still living.

This volume forms the most complete review of the subject ever presented in print and, as such, is an outstanding contribution to medicine. The papers are of a high order, and the illustrations and charts clear and adequate. There are an extensive bibliography, an index and a list of the members of the association.

Congenital Cleft Lip, Cleft Palate and Associated Nasal Deformities. By Harold Stearns Vaughan, M.D., D.D.S. 8°, cloth, 210 pp., with 259 illustrations. Philadelphia: Lea & Febiger, 1940. $4.00.

During the last few years considerable literature on cleft-palate surgery has appeared, and much progress has been made in this field. This is evidenced by the quality of the work that we see at the present time. Although some of the operations have become standardized, there is not as yet a unanimity of opinion regarding many of the pressing problems in cleft-palate surgery. In this book, the author has outlined his methods of approach based on his vast experience in this field. His main purpose is "to evaluate the various methods and to make the book of practical value to the general surgeon, to the pediatric surgeon and to the specialist in plastic and reconstructive surgery." On the whole, he has succeeded in his purpose, as the book is well illustrated with photographs and diagrams of the various operative procedures. The chapters on the primary closure of split lip and palate are dealt with more thoroughly than those on secondary repairs and late deformities in cleft-palate patients. The last two chapters dealing with orthodontic treatment and prosthetic restoration are a valuable addition, although one wishes that the subject could have been discussed in greater detail.

Whether for detailed information or for reference this book serves a very useful purpose.

Minor Surgery. By Frederick Christopher, S.B., M.D., with a foreword by Allen B. Kanavel, M.D. Fourth edition. 8°, cloth, 990 pp., with 639 illustrations. Philadelphia: W. B. Saunders Company, 1940. $10.00.

Few books are of such intense practical value to the general practitioner as this one. It emphasizes the importance of trivial injuries, and constitutes a competent and handy consultant on many of the minor conditions which so often are emergencies.

The author has recognized the difficulty of naming this work so that the title will cover adequately the many conditions described. Burns, injuries, tumors and carbuncles cannot well be lumped under any single heading. The important point is that this volume prescribes for conditions which are too often only mentioned in ordinary medical and surgical works because of their obviousness or seeming insignificance.

Some will find the editorial style difficult at first, for the varied opinions of many writers are summarized without attempt at critical analysis. But if sharp decision is somewhat lost by this method, the reader is placed in a position to choose from several methods. One physician often finds a certain therapy to work better in his hands than others; and where a choice is presented as to treatment, one form may be more readily available than another.

There is a need for all types of textbooks, but their usefulness varies widely. This one will be well thumbed by any practitioner who has it in his library.

Clinical Toxicology. By Clinton H. Thienes, M.D., Ph.D. 12°, leather, 309 pp., with 7 illustrations. Philadelphia: Lea & Febiger, 1940. $3.50.

The advent of and rapid advances in chemotherapy have focused attention on the various toxic effects of all the drugs now in daily use as well as the more uncommon ones. This small book encompasses a surprisingly large amount of material condensed in a highly utilizable form. It is evident that in the choice of material the pharmacological literature was carefully scrutinized.

The reviewer believes that a daily dose of 30 mg. of amphetamine (Benzedrine) sulfate is rather high to be regarded as within the therapeutic limit, except in some cases of narcolepsy, which today represent only a small percentage of the cases in which the drug is used. Parathormone is sometimes used in the treatment of lead poisoning; hence its toxic effects should be mentioned. Cholinergic and adrenergic are now accepted terms. The toxic effects of ethyl gasoline deserve consideration, as well as those following the administration of excessive amounts of water. Unquestionably, in future editions sulfanilamide and its related compounds will find inclusion. With these few exceptions the author has covered the subject very adequately.

Syphilis. Edited by Forest Ray Moulton, Ph.D. 4°, paper, 193 pp. Lancaster, Pennsylvania: The Science Press, 1938. $2.50.

This symposium on syphilis, presented by the Section on the Medical Sciences of the American Association for the Advancement of Science, maintains the high standard expected of this eminent association on a subject which has so recently been lifted out of its relative obscurity into the dazzling light of popularity. The contributors are all unquestioned authorities in their particular fields. The various phases of syphilis that are covered in this collection of papers are of a definitely wider range than is usually found in a monograph.

Particularly noteworthy is the obvious impartiality of the editor in presenting more than one viewpoint on certain controversial subjects. For instance, Richmond C. Holcomb's study of the Haitian history of the origin of syphilis, which he calls a "fatuous but romantic tradition," presents the thesis that syphilis is a disease of prehistoric origin with pre-Columbian existence in Europe, Asia, Polynesia and other places; this is immediately followed by William A. Pusey's "Case for the American Origin of Syphilis." Furthermore, Charles S. Butler's thesis asserts that syphilis and yaws are the same disease, while that of Howard Fox contends that they are different diseases. Ellis Herndon Hudson's fascinating story of Bejel is an important inclusion. Five different articles on the biology of *Treponema pallidum* by Ingraham, Olsen, Turner, Pearce and Beerman present a concentrated but fairly comprehensive story of the organism.

Immunity, pathology, various clinical studies, serology, chemotherapy, pharmacology and public-health aspects are presented by such an array of authorities as Michelson, Kahn, Kline, Kolmer, Solomon, Tatum, Wile and Parran, as well as others. This monograph should be on the "must list" of every syphilologist, and on the shelf of every library.